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APPENDICITIS AND ITS TREATMENT.[1]

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THIS evening we will consider the treatment of a very common disease, acute appendicitis. It is a condition that is seen by physicians, surgeons and general practitioners, and, generally speaking, throughout Australia the accepted treatment is operation immediately the condition is diagnosed. This procedure is carried even to the extreme of rushing a patient, who has been ill for days, from a warm bed, in the most inclement weather, to a hospital and operating with inadequate assistance in the early hours of the morning.

However, when we consider the results of such treatment, we find they are not so satisfactory as we would expect. We find that there has been a steady increase in the number of deaths from acute appendicitis per million of population in Great Britain, whilst in the United States of America the death rate rose 30% in the twenty-three years 1900-1922. The deaths from acute appendicitis equal all those due to salpingitis, pelvic abscess, surgical disease of the pancreas, spleen and thyreoid, gall-stones, and ectopic pregnancy combined.

In Australia the number of deaths in the years 1908-1932 has steadily increased. The death rate in a series of over 2,500 cases at Saint Thomas's Hospital, London, that is, under the best of conditions, was approximately 7%. When we consider that the people who die are mostly young and in the prime of life, we, as members of the medical profession, cannot view the present treatment of acute appendicitis with any degree of satisfaction. And the time is opportune to consider any method which will improve the outlook in a disease which, when properly treated, should have a mortality of under 1%.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 30, 1933.

It is generally accepted that the mortality from appendicitis depends directly upon the extent of the spread of inflammation when the patient is operated upon. When the disease is limited to the appendix the mortality should be under 1%. With the spread of infection beyond the appendix the mortality rate increases rapidly. It is of interest to note that the mortality rate from localized abscess is lower than the general mortality rate. It is also generally agreed that the mortality rate is greatest among those patients operated upon on the third, fourth, and fifth day of the attack. When we consider the cause of this great mortality, we find that there are two main factors.

First, it is during this period that the resistance of the patient is at its lowest ebb, but even more important is the fact that it is on the third, fourth and fifth days that the spread of inflammation from the appendix to the peritoneal cavity is progressing and is not yet walled off by the formation of adhesions, and the attack by the leucocytes on the organisms present has not yet resulted in a complete conquest of the invasion, nor has the process become localized to an abscess. Before considering the treatment of acute appendicitis we must first of all consider the pathology and the accurate diagnosis of the condition; for it is only by a proper appreciation of the pathological processes and by earlier diagnosis that we can hope to devise some treatment that will reduce the alarming mortality. The underlying change in the appendix giving rise to a dangerous attack of appendicitis is some obstruction preventing free drainage of the content of the appendix into the caecum, that is, an increased tension within the appendix.

This is brought about by the kinking of the appendix by bands, stricture of the wall, or the presence of a fæcolith within its lumen. It must be borne in mind that any inflammation of the appendix is the result of an invasion by micro-organisms which give rise to inflammation with hyperæmia and swelling, first of the mucous membrane and later of the whole appendix. This, together with the exudation into its lumen gives an increase in the appendicular content, which, owing to the factors preventing emptying of the appendix, gives rise to an increase in the pressure within its lumen, sufficient to obstruct the venous circulation. Thus a vicious circle is set up and the increase in pressure, together with the œdema of the wall, may lead eventually to a complete cessation of circulation in the appendix. The end results of these changes are: (i) The appendix may empty itself into the caecum, through the valve of Gerlach, and the inflammatory process subsides. (ii) Rupture may occur, the result of the combination of the increased appendicular pressure, infection, and the diminished circulation in its wall. (iii) Gangrene may occur, either as a true infective gangrene or as the result of the strangulation of the blood vessels, or, more commonly, a combination of the two factors.

Changes in the Peritoneal Cavity.

The presence of the inflamed appendix gives rise to inflammation in the peritoneum covering it. The reaction of the peritoneum to infection is shown by the rapid exudation of lymph. This gives rise to the turbid fluid so often seen on opening the peritoneum in these cases, and it must be appreciated that this is not pus and not a sign of infected peritonitis. It is due merely to the protective reaction of the inflammatory process. It deposits flakes of lymph about the inflamed appendix, and if perforation occurs it limits the spread of the infection to the general peritoneal cavity.

It has many times been proved that this turbid fluid is sterile and contains no dead leucocytes. The end results of these changes may be: (i) the emptying of the appendix with the resolution of the inflammatory process; (ii) a localization of the inflammatory process in the peritoneum and a resolution without abscess formation; (iii) a localization of the process with abscess formation; (iv) a spreading peritonitis.

It must be distinctly appreciated that the pathological changes in the appendix are acute and that unless the appendix has perforated or become gangrenous within thirty-six hours, perforation and gangrene are almost certain not to occur at all, unless purgatives are given, and provided that a suitable local and general treatment is carried out.

Such cases tend to subside with complete resolution. The process of localized inflammation with perforation or gangrene of the appendix is a protective mechanism. And it must be appreciated that complete resolution may occur. This can be seen clinically in the wards.

Sir James Berry says:

In one year at St. Bartholomew's Hospital, in the days when I was house surgeon and house physician, long before the time of frequent operation, thirty-one patients were admitted to the medical wards with appendicitis, or perityphlitis as it was then called. They were mostly bad cases with considerable swelling in the iliac fossa. Of these cases how many do you suppose died?

The answer is one. Hence it can be seen that patients do recover from a severe attack of acute appendicitis.

Even with localized peritonitis and with definite abscess formation, it is interesting to note that when no operation is performed there is a definite absorption of the abscess content, as it is rare to find that these patients pass pus by the bowel. Hence we see that the commonly held belief that these patients will die unless operated upon is not true; and in fact the figures quoted by Sworn and Fitzgibbon prove conclusively that the mortality is infinitely lower among patients not operated upon. In analysing the results of 487 cases of acute appendicitis, with palpable mass, Sworn and Fitzgibbon found that the mortality was 15·4% in 65 cases in which the appendix was removed and the abscess was drained, 4·03% in 124 cases in which drainage alone was done, and 0·67% in 298 cases in which no operation was done.

If complete resolution of the local peritonitis does not occur, it will probably result in the formation of a localized abscess; or if the resistance of the patient is low and the virulence of the invading organism is high, the pathological change may spread to a true generalized peritonitis, with a widespread invasion of the peritoneal cavity. This, fortunately, is not a common occurrence, and the more one sees of these cases, the more patient and conservative one becomes. Even if the complete resolution of the process cannot always be obtained, localization of the process to a free abscess which requires only a small incision for drainage without involving the peritoneal cavity, gives results very much more satisfactory than would be obtained by earlier operation.

Pathological Changes in Relation to Symptoms.

We are now in a position to appreciate the pathological processes underlying the symptoms of acute appendicitis.

1. Central abdominal pain is a referred pain, the result of the increased tension in the appendix. It is sudden in onset and constant, with perhaps some colicky exacerbations, and persists as long as the appendix is distended. It ceases with the emptying of the appendix through the valve of Gerlach or the rupture or gangrene of the appendix. Umbilical pain is the cardinal symptom of those cases of acute appendicitis which give rise to perforation or gangrene, that is to say, those cases with some obstruction in the appendix. It is conceivable that in very rare circumstances, such as the perforation of the appendix by a pin, acute perforative appendicitis can occur without tension in the appendix, and the resultant typical umbilical pain. When there is a simple inflammation of the appendix with no obstruction, umbilical pain is not necessarily present, but it is these cases which do not give rise to localized peritonitis, and the mortality is under 1%. Hence we see that in the dangerous case of appendicitis umbilical pain is the most constant symptom. I have carefully inquired for many years for this symptom and it has always been present. The acute onset of the pain is closely followed by anorexia, nausea and perhaps vomiting of the stomach content only.

2. With the spread of the inflammation to the peritoneum covering the appendix, the localizing sign of tenderness and rigidity in the right iliac fossa or in the position in the abdominal cavity in which the appendix is situated, occurs. Unfortunately, pain in the right iliac fossa is looked upon by the patient, and sometimes by the physician, as the cardinal symptom of appendicitis, but all that this means is the presence of an inflammatory process in the peritoneal cavity. In acute appendicitis the pain may be low down in the pelvis, high up in the loin, or even, as I have seen it, below the left costal margin, depending upon the position of the appendix.

3. Another sign of great importance is that of hyperæsthesia, denoting distension of the appendix. It is constantly present in the region of Poupart's ligament and in the thigh just below it, that is, along the distribution of the twelfth dorsal and the first lumbar nerves. This hyperæsthesia must not be confused with a hyperæsthesia above Poupart's ligament, when an abscess has formed under pressure. The combination of prolonged diffuse central abdominal pain, starting usually in the early morning, followed by nausea and perhaps vomiting, and later settling down as a localized tenderness and rigidity in the right iliac fossa, is a clinical picture so typical and so constant that no intelligent practitioner should wait longer before advising removal of the appendix.

Treatment.

The treatment of acute appendicitis is removal of the appendix. There is no serious objection to this principle. For it is well established that once the appendix becomes affected by a pathological change that gives rise to acute inflammation, further attacks will follow.

Some years ago it was taught that once an appendiceal abscess was drained it was not necessary later to remove the appendix, as further attacks did not occur. This is a fallacy which every surgeon of experience can expose. It is unusual, however, to have a severe recurrence of acute appendicitis within three months of a previous acute attack. Furthermore, it has never been proved that the appendix has any useful function, and experience has taught us that no derangement of function follows its removal. I think, therefore, that all surgeons will agree that once a patient has an acute attack of appendicitis, the appendix should be removed. The next point to be considered is the time of removal. The generally established custom of treating every case of acute appendicitis by immediate operation when the patient is first seen has given results which have an abnormally high mortality rate of about 7%. This is a position which, when duly considered, should not be looked upon with any great pride, and it is only by earlier diagnosis and a more careful selection of the time of operation that our results will improve.

We know:

1. That when the inflammatory process is limited to the appendix the mortality is less than 1%.

2. That when the appendix is removed between acute attacks the mortality is about 0·5%.

3. That with the spread of inflammation beyond the appendix into the peritoneal cavity the mortality of the operation rises rapidly on the third and fourth days and then falls on succeeding days. It is during the second to the fifth day that the protective mechanism of the peritoneum comes into action and limits the spread of inflammation and the local and general resistance of the patient is at its lowest ebb.

4. That under proper medical treatment, carefully supervised and carried out, the peritoneum can deal with a perforated or gangrenous appendix, limit the spread of true peritonitis and lead

TABLE I.

Patients operated on within twenty-four hours of onset.

Group.	London Hospital.	St. Thomas's Hospital.
Number of cases	221	303
Recovered	219	296
Died	2	7
Mortality *per centum*	0·9	2·3

eventually to a complete resolution with no adhesions in the peritoneal cavity.

It can be laid down that when the inflammatory process is confined to the appendix, immediate operation for its removal should be undertaken. In arriving at the conclusion that the appendix is intact, the following are the important criteria:

1. Umbilical pain is still present.
2. Hyperæsthesia in the region of Poupart's ligament and below is still present.
3. As a general rule it may be said that if the patient is seen within twenty-four hours of the attack the appendix will still be intact.

If, however, the patient is seen after thirty-six hours of the onset of the attack, acute appendicitis is no longer to be treated as a surgical emergency. The patient is placed in a strict Fowler's position, no purgative or enema should be given, water by the mouth should alone be allowed, unless there is marked vomiting, when saline solution may be administered by the rectum, and the pain should be relieved by hot flannel foments applied to the abdomen. A four-hourly chart should be kept of the pulse and temperature, and the patient should be carefully watched. The progress of the abdominal condition, whether rigidity, tenderness or tumour formation be present, should be determined frequently. Under this treatment it will be found that the temperature and pulse rate will fall to normal, the tenderness and rigidity in the abdomen will gradually become less and more localized, and the mass, if present, will become smaller and eventually disappear. The pain will gradually lessen and the appetite return, and in the vast majority of cases the conditions of the peritoneum and the appendix will return to normal so that when the operation is undertaken later no lesion of the appendix, apart from a little dilatation and flabbiness, will be found. The astounding fact is that no gross adhesions will be found in the peritoneum, even after a known intraperitoneal abscess. Occasionally such a complete resolution in the pathological process does not occur, whether it be a generalized or local peritonitis or a local abscess, but even amongst these failures it must be appreciated that a delayed treatment has converted many cases of general or local peritonitis to a local abscess, a condition with a much lower mortality. Hence it must be laid down that if under the strict adoption of the treatment prescribed the temperature and pulse show no fall, or if a patient complains of increasing pain, or the process in the abdomen is spreading, operation will have to be undertaken. During conservative treatment repeated rectal examinations should be undertaken to determine the presence of a pelvic collection, signs of which are mucous diarrhœa and the dilatation of the anal sphincter.

TABLE II.

Patients treated on delayed lines.

Group.	London Hospital.	St. Thomas's Hospital.
Inflammation subsided:		
Number of cases	232	143
Recovered	227	143
Died	5	0
Mortality *per centum*	2·1	0·0
Unsuccessfully delayed:		
Number of cases	109	71
Recovered	102	62
Died	7	9
Mortality *per centum*	6·4	14·0
Total:		
Number of cases	341	214
Recovered	329	205
Died	12	9
Mortality *per centum*	3·5	4·2

TABLE III.

Cases in which immediate operation was performed.

Group.	London Hospital.	St. Thomas's Hospital.
Inflammation limited to the appendix:		
Number of cases	633	348
Recovered	627	345
Died	6	3
Mortality *per centum*	0·9	0·8
Local peritonitis:		
Number of cases	466	271
Recovered	438	257
Died	29	14
Mortality *per centum*	6·2	5·2
Local abscess:		
Number of cases	347	95
Recovered	331	91
Died	16	4
Mortality *per centum*	4·6	4·2
General peritonitis:		
Number of cases	230	187
Recovered	183	132
Died	47	55
Mortality *per centum*	20·5	29·4
Total:		
Number of cases	1,677	901
Recovered	1,579	825
Died	98	76
Mortality *per centum*	5·8	8·4

TABLE IV.

General Peritonitis Cases. (All treated by immediate operation.)

Day of Attack.	London Hospital. (230 Cases.) (Percentage.)	St. Thomas's Hospital. (187 Cases.) (Percentage.)
First	6·6	10·6
Second	29·8	46·5
Third	35·6	21·3
Fourth	13·3	9·0
Fifth	6·6	5·8
Sixth	4·7	1·0
Seventh	1·1	3·7

TABLE V.
Mortality of operation on the different days of the attack.

Number of Days.	Inflammation Limited to Appendix.		Local Peritonitis.		Local Abscess.		General Peritonitis.		Total.		Mortality Percentage.
	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	Recovered.	Died.	
1	191	1	52	1	20	0	16	1	279	3	1·1
2	264	2	61	2	45	2	69	7	439	13	2·8
3	82	2	79	12	123	8	72	19	356	41	10·3
4	21	0	67	9	71	5	26	8	185	22	10·6
5	8	0	22	2	29	0	12	5	71	7	8·9
6	10	1	31	0	32	1	9	3	82	5	5·7
7	4	0	37	1	11	0	2	1	54	2	3·6
8	7	0	19	1	9	0	1	0	36	1	2·7
9	31	0	32	1	15	0	0	0	78	1	1·2
10 (or more)	133	1	88	4	57	1	3	1	281	7	2·4
Total	751	7	488	33	412	17	210	45	1,861	102	5·2

Sherren, in an address in 1923, said:

In the first paper that I published on the treatment of acute appendicitis in 1905, I wrote of these patients that they should be tided over the attack if possible and the appendix removed later. This has been my rule in the wards of the London Hospital, and nearly twenty years' experience has only served to strengthen the opinion that this is the best treatment .... The only change I have made has been to greater conservativeness and more patience in dealing with cases of appendix abscess.

Types of Cases for Operation.

We must consider three types of cases for operation.

1. *Operation on the Intact Appendix within the First Twenty-Four Hours.*—The best method of approach is undoubtedly by the muscle-splitting incision of McBurnie, and the appendix is removed in the usual manner. It is unusual to find the necessity for drainage in these cases, and a very good rule is the old adage, "when in doubt, don't". But occasionally a large collection of fluid is found, extending down into the pelvis, and then the best method of drainage is by a separate suprapubic stab incision, with the tube running down well into the pouch of Douglas, otherwise a simple cigarette drain through the muscular part of the abdominal wall is all that is necessary.

2. *Cases in which the Conservative Treatment has Failed.*—(*a*) In cases of general peritonitis the best method of approach is through a long McBurnie's incision. In these cases it is essential that the appendix be removed and the peritoneal cavity drained through a suprapubic drain incision. (*b*) In cases of local abscess formation in the iliac fossa a small muscle-splitting incision is made as far as possible from the unaffected peritoneal cavity. The abscess cavity is opened by a finger or forceps, and a large tube is inserted into the cavity. No attempt should be made to remove the appendix. (*c*) In cases of a pelvis abscess, undoubtedly the best method of approach is through the rectum. The patient is placed in the lithotomy position, a retractor is placed along the anterior wall of the rectum, the bulging rectal wall should be incised transversely and a tube inserted in the abscess cavity and fixed to the anal margin. Even in women this method gives better results than drainage through the posterior vaginal fornix.

3. *Those Cases in which the Inflammatory Process has Subsided.*—When the inflammatory process has subsided a McBurnie incision is quite adequate, but if a general exploration is required, a right lower paramedian incision is best, as it can be enlarged to any extent without affecting the abdominal wall. The question in these cases is to decide on the best time to remove the appendix after an acute attack, that is, when the inflammatory reaction has subsided and the adhesions in the peritoneal cavity have been absorbed, but before a further acute attack supervenes. From experience it is found that three months after the acute attack is the best time for the removal of the appendix.

Post-Operative Treatment.

The post-operative treatment of all classes of patients is essentially the same. As soon as the patient recovers consciousness he should be placed in Fowler's position; fluid should be administered as soon as possible by the mouth. If necessary, saline solution and glucose should be given by the rectum. Purgatives should at all times be avoided until the bowel has been opened by an enema forty-eight hours after the operation. Morphine can be given in sufficient doses to relieve pain, but repeated doses are not to be encouraged.

The question of drainage tubes is one which has undergone a considerable change in recent years. It must be appreciated that the track of a tube is probably sealed off by adhesions quite soon after its insertion, and no point is gained by retention of a tube after thirty-six hours, whilst there are many disadvantages.

Conclusion.

I fully appreciate the fact that the treatment that I have advocated tonight will not meet with the approval of you all. Some of you practise it, some of you approve of it in theory but do not practise it, and some will frankly condemn it. I can assure you that I was as antagonistic to it as any of you until I was working in a hospital in

London and saw the results obtained by surgeons who insisted on an immediate operation, and the results of those who treated their patients on the lines of which I have spoken tonight. Looking back over the experience of many hundreds of cases of acute appendicitis, I know that I have no regrets of having used conservative treatment. But I very sincerely regret that I have operated on some patients who would have been better left alone. All of you must recall in your own experience some stalwart young man in the prime of life who walked into your consulting room, complaining of some abdominal pain, but not at all critically ill. You have found a mass in the right iliac fossa and given a good prognosis and advised immediate operation. The operation has been skilfully performed, but the patient has become critically ill and died. Such a patient would probably have recovered if no operation had been undertaken.

Attention is directed to the tables accompanying this article.

CAUSES OF BLINDNESS.[1]

By SIR JAMES BARRETT,
Melbourne.

AT the Australasian Medical Congress held in Melbourne in 1923 I presented the results of an analysis of 192 applications for admission to the Royal Victorian Institute for the Blind for the period 1901 to 1923. The result is reproduced in Table I.

TABLE I.

Disease.	Number of Persons.	Percentage of Total. (Approximate.)
Optic atrophy	47	25·0
Chorioiditis	21	11·0
Ophthalmia neonatorum	21	11·0
Corneal ulceration	15	8·0
Injury	14	7·0
Trachoma	13	6·7
Nystagmus	10	5·2
Cataract	9	5·0
Buphthalmos	9	5·0
Degeneration of vitreous	5	2·5
Shrunken globe	5	2·5
Kerato-iritis	4	2·0
Retinitis pigmentosa	4	2·0
Doubtful	2	1·0
Interstitial keratitis	3	1·5
Cerebro-spinal meningitis	2	1·0
Glioma	2	1·0
Microphthalmos	2	1·0
Panophthalmitis	1	—
Myopia	1	—
Retinal hæmorrhage	1	—
Degeneration of the cornea	1	—
Total all diseases	192	

I have now to present the analysis of patients seeking admission for the last ten years. The analysis is difficult because in many cases no history is available, but Dr. Parnell has made a thorough investigation of these cases as far as was practicable. On her notes, including such cases as are available, the summary is based.

[1]The President's Address read at the Annual Meeting of the Ophthalmological Section of the Victorian Branch of the British Medical Association on November 30, 1933.

In this instance I have divided the patients into two groups, those under and those over fifteen years of age, as there is much to be learned by such separation (see Tables II and III).

TABLE II.

Applicants admitted to the Royal Victorian Institute for the Blind who were under the age of fifteen years from November, 1923, to November, 1933.

Causes of Defective Vision.	Number of Cases.	Percentage of Total. (Approximate.)
Optic atrophy	13	14·0
Myopia	11	12·0
Nystagmus	10	11·0
Ophthalmia neonatorum	9	9·5
Interstitial keratitis	7	7·5
Degeneration of vitreous	5	5·0
Albinos	5	5·0
Chorioiditis	4	4·0
Congenital cataract	4	4·0
Leucoma	3	3·0
Sympathetic ophthalmitis	3	3·0
Trachoma	2	—
Shrunken globe	3	—
Iritis	3	—
Aniridia	2	—
Retinitis pigmentosa	1	—
Macula degeneration	1	—
Microphthalmos	1	—
Kerato-iritis	1	—
Post polar cataract	1	—
Injury	1	—
Hypermetropia, high-grade	1	—
Buphthalmos	1	—
Dislocation lens	1	—
Glaucoma	1	—
Total cases	94	

Of rejected applicants no less than thirteen possessed good vision ranging from $^6/_5$ in both eyes to $^6/_{12}$ partly. In addition, a large number possessed vision of $^6/_{36}$ to $^6/_{18}$, and in most cases with fairly normal fields and with no condition that incapacitated them. They sought to enter the institution because of the relatively easy work and certainty (as they thought) of getting at least the basic wage. It is the practice of the institution to pay the difference between the money they earn and the basic wage. It is a practice based on abstract justice, and applicable to the blind or the partially blind.

Total blindness is defined as inability to count fingers at one metre. Partial blindness is vision of $^6/_{60}$ or less.

For those over $^6/_{60}$ each case is considered on its merits. If contracted fields or nystagmus are present they are taken into account as well as the general mental condition.

Retinitis pigmentosa with contracted fields and vision of $^6/_{24}$, or even more, may be admitted. I have under my care, however, an albino with nystagmus and vision of $^6/_{60}$ in each eye who is practically managing and working a farm with success.

The blind and the partially blind require different economic treatment, but the partially blind object and state that the sight they possess is a drawback to their work.

In all such organizations human nature operates powerfully. It is obvious that without strict medical

control the institution would be flooded with people quite able to earn their own living so far as sight is concerned.

Patients Under Fifteen Years of Age.

Optic Atrophy and Syphilis.

Of the thirteen cases of optic atrophy four were the result, apparently, of intracranial conditions and optic neuritis, nine were definitely syphilitic in the majority of cases, and in all syphilis was almost certainly the cause. If to these nine be added the seven cases of interstitial keratitis, the four cases of chorioiditis, the case of kerato-iritis, and the five cases of vitreous degeneration, the total number of cases due to syphilis is twenty-six. But, in all probability, to this number must be certainly added some of the cases of nystagmus and of congenital cataract and iritis. In some of these there was direct evidence, in others only supposition. The reason for including the cases of congenital cataract is the fact that though the patients were operated on by competent surgeons, the result was unsatisfactory.

Venereal Disease.

If to the certain syphilitic cases be added the nine cases of *ophthalmia neonatorum*, at least thirty-five of the ninety-four cases are due to venereal disease. The patient with macular degeneration had a sister suffering from interstitial keratitis.

Congenital Defects.

On the other hand, the congenital anomalies are numerous, namely *retinitis pigmentosa*, dislocation of the lens, aniridia, hypermetropia, buphthalmos, albinism and microphthalmos.

Nystagmus.

Many of the patients showed nystagmus, but the ten patients set out as manifesting nystagmus did not show any lesion that accounted for very defective vision.

Trachoma.

It is noteworthy that only two cases of trachoma were presented in ten years.

Myopia.

Myopia assumes a serious aspect. The grade of myopia, in diopters, and the ages are set out as follows:

	Diopters.	Diopters.
7 years	−13 and	−15
13 years	−17 and	−15
14 years	−11 and	−10
10 years	−15 and	−14
12 years	−17 and	−8
11 years	−15 and	−13
3½ years	−15 right and	left
6 years	−10 and	−9
3 years	−2·5 and	−4
8 years	−3 and	−1

It may well be asked why should a child of three and a half years suffer from myopia of fifteen diopters in each eye? The association of one of the cases with interstitial keratitis is significant.

Patients Over Fifteen Years of Age.

TABLE III.

Applicants admitted to the Royal Victorian Institute for the Blind who were over fifteen years of age from November, 1923, to November, 1933.

Cause of Defective Vision.	Number of Cases.	Percentage of Total. (Approximate.)
Myopia	20	15·0
Optic atrophy	17	13·0
Trachoma	12	9·0
Retinitis pigmentosa	11	8·0
Chorioiditis	8	6·0
Nystagmus	7	5·0
Interstitial keratitis	6	4·5
Injury	4	3·0
Iritis	4	3·0
Detachment retina	4	3·0
Congenital cataract	4	3·0
Shrunken globe	4	3·0
Buphthalmos	4	3·0
Amplyopia	4	3·0
Sympathetic ophthalmitis	3	2·0
Ophthalmia neonatorum	3	2·0
Congenital dislocation of lens	3	2·0
Vitreous degeneration	3	2·0
Leucoma	2	—
Retino-chorioiditis	1	—
Coloboma disk	1	—
Lime burn	1	—
Macular degeneration	1	—
Leber's disease	1	—
Glaucoma	1	—
Retinal hæmorrhage	1	—
Albino	1	—
Kerato-iritis	1	—
Total cases	132	

Optic Atrophy and Venereal Disease.

Of the seventeen cases of optic atrophy two were not syphilitic; several patients gave positive Wassermann reactions, and it may be assumed that fifteen cases were syphilitic in origin. The same assumption may be applied to the three cases of vitreous degeneration, the five cases of iritis, the case of kerato-iritis, the six cases of interstitial keratitis and the eight cases of chorioiditis.

Several were definitely syphilitic, and as the other cases were similar in character, the thirty-seven cases may be set down as syphilitic. But one patient with nystagmus also gave a positive reaction.

If to these be added the case of *ophthalmia neonatorum*, it may be safe to ascribe forty cases to venereal disease.

Nystagmus.

Cases of nystagmus remain an enigma, as do the cases of amblyopia. The nystagmus patients showed no evidence of any organic cause of blindness. But in young and old nystagmus was the only objective evidence. Whether any of them would show lesions in the retino-cerebral apparatus remains to be ascertained. These remarks apply equally to the amblyopic patients. The two conditions account for eleven cases.

Trachoma.

Whilst there were only two patients with trachoma in ten years under the age of fifteen, one of whom has recovered, there were twelve disastrous cases among patients over the age of fifteen. Of course, cases under fifteen may go to the bad later in life, but, nevertheless, the figures give some indication of the way in which trachoma is disappearing from Victoria. New cases in Australians are now uncommon.

Myopia.

Myopia heads the list as the most common cause of blindness in people over fifteen years of age. Combined with its high grade in those under fifteen years of age, it obviously requires more attention than it has received. The amounts of the defect in some of the cases are here set out.

Ages.	Diopters.	Diopters.
26 years		−15 and −18
20 years		−20 and −25
20 years		−25 and −25
52 years		−22 and −25
19 years		−16
18 years		−22
32 years		−25
49 years		−31 and −26

Some of the patients showed nystagmus, and some cases were also associated with signs of degeneracy and two cases with syphilis, as indicated by interstitial keratitis and chorioiditis.

It is obvious that myopia is a definitely progressive disease in many cases, depending on general nutritive disturbance and not necessarily associated with the use of the eyes for near work.

Retinitis Pigmentosa.

Eleven cases of *retinitis pigmentosa* make a disquieting item. One was associated with deafness. It is clear that if these patients are allowed to rear families there is no hope of eliminating this tragic disaster.

Sympathetic Ophthalmitis.

Sympathetic ophthalmitis accounts for only three cases in ten years; these, together with three cases in patients under the age of fifteen, make six cases in all.

Patients with Good Vision.

As in the case of those under fifteen years, many people with excellent vision sought admission for economic reasons. Particulars of some cases are set out.

Ages.	Vision.
58 years	$^6/_6$ and $^6/_6$
34 years	$^6/_{12}$ right and left
19 years	$^6/_{12}$ and $^6/_{18}$
45 years	$^6/_6$
19 years	$^6/_{12}$
50 years	$^6/_{12}$
33 years	$^6/_9$
34 years	$^6/_{12}$

There are about 1,000 blind people in Victoria, so that the total cases analysed represent about two-fifths of the total. There are no means of ascertaining the causes of blindness in the other three-fifths. But the cases analysed give a good indication of causation subject to certain reservations.

Cases of glaucoma and some cases of detachment of the retina occur late in life, and such people are too old or ill to take advantage of the methods of education provided at the institution. Some cases of myopia also fall under this category. Many of the patients of all kinds who sought admission are degenerates, and require institutional treatment.

Many Wassermann tests were made, but in many cases it was not possible to make the test. In the following and most other instances there was definite proof of syphilis: one case of macular change, one case of degeneration of the vitreous, two cases of kerato-iritis, one case of nystagmus, one case of trachoma, one case of chorioiditis, one case of iritis, and several cases of optic atrophy.

TABLE IV.

Total number of cases—all ages.

Disease.	Number of Cases.	Percentage of Total. (Approximate.)
Myopia	31	13·5
Optic atrophy	30	13·0
Nystagmus	17	7·5
Trachoma	14	6·0
Interstitial keratitis	13	6·0
Ophthalmia neonatorum	12	5·5
Retinitis pigmentosa	12	5·5
Chorioiditis	12	5·5
Congenital cataract	8	3·5
Vitreous degeneration	8	3·5
Iritis	7	3·0
Shrunken globe	7	3·0
Sympathetic	6	2·5
Albino	6	2·5
Injury	5	2·0
Buphthalmos	5	2·0
Leucoma	5	2·0
Detachment retina	4	—
Congenital dislocation lens	4	—
Amblyopia	4	—
Kerato-iritis	2	—
Macular degeneration	2	—
Aniridia	2	—
Glaucoma	2	—
Retino-chorioiditis	1	—
Coloboma disk	1	—
Lime burn	1	—
Leber's disease	1	—
Retinal hæmorrhage	1	—
Microphthalmos	1	—
Post polar cataract	1	—
Hypermetropia	1	—
Total cases	226	

Comparison with the Record of 1901-1923.

It is noteworthy that in the list of trachoma cases prior to 1923 the average age of the patients was twenty-three, and the youngest was eleven years of age. Cases of trachoma in young people are now uncommon, and it is probable that the cases seen in people over fifteen years of age during the last ten years really represent the residual cases of a disease which is rapidly disappearing in Australians.

It is evident that a definite change has occurred in the conditions causing blindness. *Ophthalmia neonatorum* and trachoma have definitely diminished. Optic atrophy is not so frequently seen. But myopia has made its appearance as a major cause of blindness, and *retinitis pigmentosa* has definitely increased.

In 1923 I estimated that 50% of the cases were due to some form of venereal disease, and I am inclined to think that proportion is now a little less, owing probably to the increase in number of the cases of myopia. The percentage is in all probability between 40% and 50%.

Remedies.

There are apparently only two directions in which substantial reduction may be effected. If venereal disease were eliminated, at least 30% of the cases would not exist. I think that this estimate is

conservative and that the percentage would be much higher. But that is a matter of speculation. Furthermore, it is impossible to prove that much of the degeneration is remotely syphilitic in origin, but yet the evidence is strong enough to prevent the theory being laid aside. As in so many cases of advanced and hereditary syphilis, the Wassermann results are negative.

Myopia must be surveyed from a new viewpoint, and such cases as those presented must be regarded, not as errors of refraction, but as cases of a malignant uncontrollable disease. The numerous cases of *retinitis pigmentosa* and the myopic cases open up the eugenic problem in an acute form.

A SALMONELLA INFECTION (BACILLUS TYPHI MURIUM) IN A STOCK OF EXPERIMENTAL MICE, WITH OBSERVATIONS ON THE MORBID ANATOMY AND EPIDEMIOLOGY.

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AT the middle of the summer of 1931-1932 there occurred in mice at the University Cancer Research Laboratory several deaths in isolated cages. From the same cause deaths continued to occur for five months to a total of six hundred.

These cases were investigated with regard to causative organism, morbid anatomy and some factors relative to the epidemic cycle. The last mentioned cannot be considered as conclusive because the epidemic was an incident in another experiment, not a planned epidemiological experiment.

Bacteriology.

From the liver, stomach, heart blood and spleen of these mice a Gram-negative bacillus was isolated. It grows readily on agar, but more readily on serum agar, produces in twenty-four hours a smooth opalescent culture which becomes white and opaque, but does not roughen within four weeks; it grows readily in beef infusion broth, and ferments glucose with formation of acid and gas, but does not affect lactose or saccharose; it reduces Rochelle salt in tartarate medium; it is actively motile in fresh culture and measures 1·5μ by 0·4μ when stained, that is, it is a member of the Salmonella group. As no reliable sera for agglutination were to hand, an estimation of its group titre was not done.

One cubic centimetre of a forty-eight hour beef infusion broth culture fed individually to mice in bread and bran killed the mice in three to five days. To eliminate the possibility that the cause of death was the type of toxin investigated by Savage and White,[1] Menten,[2] Branham, Robey and Day,[3] bacilli from twenty-four hour agar plate cultures were washed twice in saline solution. The emulsified deposit was fed in a small amount of bran and bread to five isolated mice. These were from the same litter, four months old; the dosage was the same for each. Death occurred in five to nine days. Pure cultures of the organism were obtained from the liver, spleen and heart blood, which reproduced the condition in other mice to which they were fed. The cultural reactions and the pathogenicity of the organism prove its identity with *Bacillus typhi murium*.

In observing the temperature of mice infected with this organism, five mice were examined by seven daily rectal thermometer readings. They showed individual variation of 1° to 2° C. and some fluctuation with variation of external temperature. They were then fed with washed bacilli as above. The temperature in each case rose on the second day 1° to 2° C., but two to three days before death commenced to fall, and though the animal was not obviously very ill, reached 4° C. below normal, at which it continued until the animal became moribund and died.

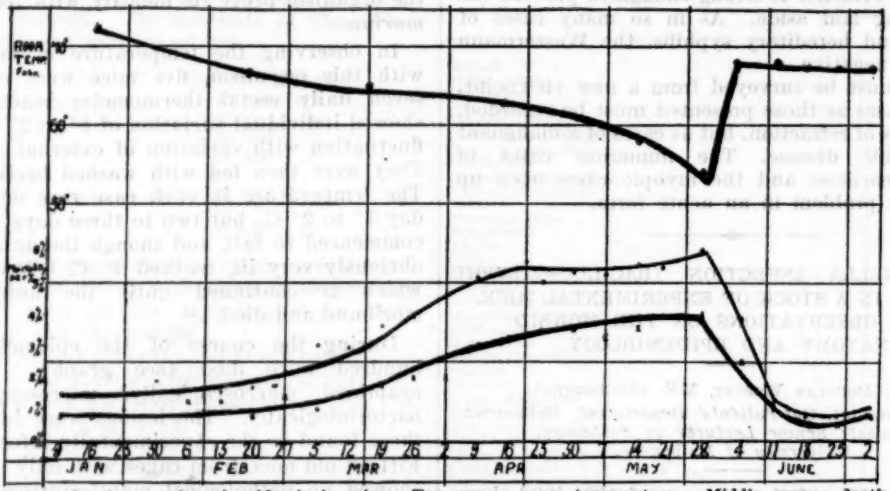
During the course of the epidemic some six hundred mice died (see graph). These were examined macroscopically, microscopically and bacteriologically. The lesions were identical with those found in the experimentally infected animals. Fifteen old mice from cages with only one occupant showed no pathological manifestations of disease. Ninety-eight adult mice, which presented no evidence of acute infection, classified separately, from infected cages died during and subsequent to the epidemic. Fifty of these showed evidence in the liver and stomach of previous infection. These lesions will be discussed in the following section.

Morbid Anatomy.

In the cases of mice dying of acute infection, the alimentary canal presents a series of lesions. In the stomach superficial ulceration in discrete small areas is common. This also occurs in the first part of the duodenum (Figure I). There is a local necrosis and desquamation of epithelium, endothelioid, polymorphonuclear and round cells are in excess of normal, fibrin is present in the *tunica propria* in association with myriads of bacteria; gradations can be traced from these lesions to the chronic lesions observed in the fifty mice dying without evidence of acute infection. In these mice, at the pylorus, more often on the duodenal side in the Brunner's gland area, there occur ulcers with a superficial necrotic tissue overlying a well formed fibrous granulation tissue base. Round cells with a large amount of protoplasm and pyknotic nuclei are frequent in the tissue (Figure II). Organisms could not be stained in these ulcers, but could be readily demonstrated in the less chronic ulcers which had apparently formed from previously acute lesions. These ulcers are worthy of note because of the extreme difficulty of experimentally producing chronic ulcers of the human "peptic" type.

The experiment which was being undertaken at the time of the epidemic involved the intradermal injection of minimal doses of tubercle bacilli by Cherry's method. In the mice in which no such injection had been made the ulcer was of the simple granulation tissue type. In some of the mice

injected with tubercle bacilli epithelial hyperplasia occurred in the base of the ulcer.[10] The small intestine was not examined in all cases. In those examined (54) it showed no definite lesion.



Graph showing results of epidemic in mice. Top curve: room temperature. Middle curve: death rate in 450 mice aged under 3 months (total deaths 313). Bottom curve: death rate in 700 adult mice (over 3 months) (total deaths 280). At the end of May the change in *régime* detailed above was instituted.

The caecum in the acute cases shows uniformly a reddened peritoneal coat, and microscopically ulcers of the type described in the stomach, that is,

FIGURE I.
The acute lesion of the post-pyloric part of the duodenum is shown. Destruction of the surface epithelium and early inflammation are seen. The freely movable pancreas has become adherent to the base of the ulcer. × 80.

desquamation and acute degenerative inflammatory reaction with many bacilli.

The pancreas in the acute cases presents a picture of vascularity, œdema separating the pancreatic acini with diffuse and intense leucocytic infiltration. In the chronic cases the dorsal pancreas has usually become normal with resolution of the inflammation. In some cases, however, it is represented only by fibrous and fatty tissue enclosing sequestered pancreatic islets. The ventral pancreas is more often degenerated, sometimes because the duct is involved in the duodenal ulcerative process, sometimes from the inflammatory reaction. In the former case cysts and dilated remnants of acini may be seen with healthy islet tissue incorporated in the mass; in the latter the islet tissue is segregated in a fibro-fatty matrix.

The liver in the acute process presents either focal lesions in the adult mice (aged three months and over) or a generalized lesion, which occurs more frequently in the young mice. The focal lesions present a central necrotic mass of liver cells with bacteria surrounded by endothelial cells and leucocytes. They may be seen as small distinct or large necrotic white areas macroscopically. In the generalized condition the liver cells are cloudy and the sinusoids crowded with leucocytes; bacteria occur throughout. It simulates closely in histological appearance leuchæmic infiltration of the liver. In the chronic condition, which may or may not be associated with pyloric or pancreatic disease, these focal areas are replaced by areas of hyaline fibrous tissue.

The lungs of 157 of the acutely infected mice also present lesions. They are visible macroscopically as whitened areas one to two millimetres in diameter. Microscopically these have a centre of necrotic material with many organisms, surrounding congestion, œdema with cellular and fluid exudation. No resulting chronic lesions were observed. Three cases of focal suppurative myocarditis occurred.

These manifestations, though occurring in the majority of the animals that died of the epidemic,

were not present in all. In the young mice especially one had to rely on liver and spleen culture.

An epidemic of *pseudotuberculosis murium* and *rodentium* had previously occurred in this laboratory. The lesions are somewhat similar to those here recorded, but the small intestine is uniformly affected, the liver lesions are more opaque, white and discrete, the cultural reactions and morphology of the organism are distinct. Only two cases occurred this year, one during the epidemic in an isolated cage, one after the epidemic came under control.

kept on benches in a room devoid of any temperature-regulating device. Breeding was allowed to go on, the young to three weeks of age being transferred with their nest to a new tin; after that period a new nest was provided. If they are shifted from the nest earlier the mother is liable to abandon them.

Thus four factors influencing the course of the epidemic were present. They bear directly on the conclusions drawn from the results of experimental epidemics by Topley and his colleagues.[4][5][6][7][8]

The first factor was the presence in the tin of infected mice for a week along with other mice

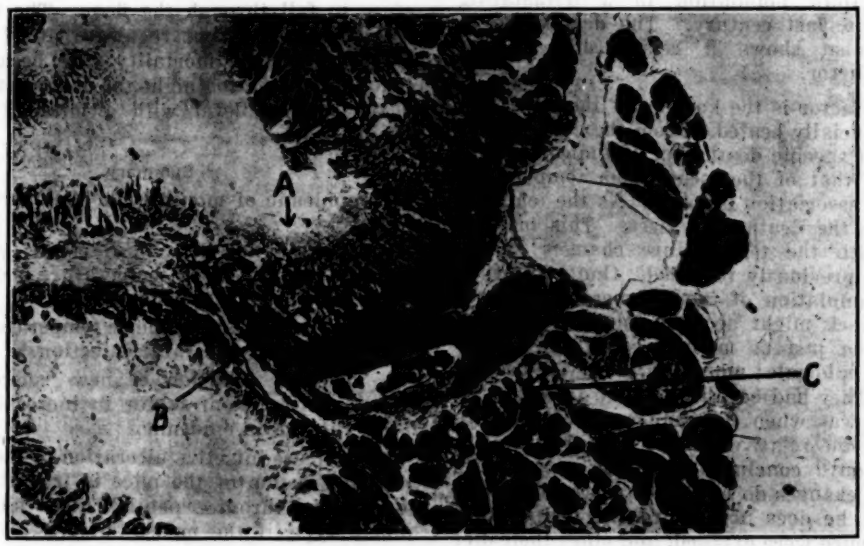


FIGURE II.
A chronic ulcer is seen with necrotic material lying on the base (A), granulation tissue with extensive round-celled infiltration (B), and adhesion of the pancreas (C). The splenic vein and artery are implicated in the adhesion. × 8.

Epidemiology.

We come now to consider the epidemiological characteristics of this infection, the factors which facilitated its spread and affected the total mortality.

The mice were at the time kept in petrol tins. Each tin had a side removed and was closed with a wire mesh lid. About ten to twelve mice per tin was the average population. "Hygienic Flock" was provided for bedding at one end of the tin, but not in a compartment separated from the remaining floor space, which was covered with sawdust fresh from the blowers of the saw benches. This was necessary to avoid the contamination of the sawdust by cat's fæces which occurs in the sawdust dumps. Cysticercus infection was thus avoided. Food of varied type, adequate in vitamin, caloric and salt value, was introduced daily. Water was provided by an inverted bottle with a glass tube mouthpiece. Each week the mice were transferred manually to the new, clean, sterilized tins and the soiled cages were cleaned out and sterilized. They were then without transfer or cleaning. Thus cross infection had every chance of taking place. But the disease was not as uniformly fatal as when the bacteria are fed specifically to hungry animals (*vide* feeding experiments above). Deaths occurred in the affected tins occasionally, rising over the whole series to a maximum associated with the fourth factor (see graph). The presence of carrier mice must be assumed from Topley's work. The alimentary canal lesions here noted may explain this.

The second factor was the manual removal of the stock. Thus sixty tins of mice were infected, as judged by specific deaths before attempts were made to isolate them. These were already apparently infected when the death rates were first calculated. That it affected the mortality curve is unlikely, since the stock of a cage in which there has been infection is always at risk.

The third factor which bears on Topley's observations and deductions was the continuance of breeding in the tins. The young as a rule remained healthy for three or four weeks, that is, until they

left the nest, which mice rarely foul when there are young in it, and commenced to feed round the cage with the adults. Deaths then began to occur at a rate approximately twice that occurring in the adult mice. This agrees with the observation of Greenwood, Newbold, Topley and Wilson[8] that there is a maximum death rate in susceptible mice newly added to an infected stock at approximately forty days after addition. The previous experience of their mice is not, however, known. Their results and mine may be compared, this with the infection of young children with measles in civilized communities and that with the infection of the adult susceptible Fijian population in a devastating epidemic in the last century. The death rate in these mice also shows a relationship to the temperature factor.

The fourth factor is the keeping of the mice in a room not artificially heated. From the curve of the weekly rate of specific deaths per 100 mice (weekly average) and that of the mean room temperature a remarkable association is found. As the temperature falls, so the death rate rises. This may be associated with the temperature changes in the infected mice previously recorded. One may argue that the accumulation of the summer breeding of susceptible stock might account for this rise, but there had been just as many bred in the earlier stages of the epidemic; more had, however, become adult mice. They had been exposed to infection all the time. It was when the change of temperature occurred that the relatively rapid rise of mortality set in. Wilson[9] concluded from his experiments that seasonal changes do not affect susceptibility to infection, but he does not record the temperature to which his mice were exposed, and since their diet was unbalanced and their previous bacteriological history unknown, it is uncertain what influence these factors had on the death rate of his mice. Until one knows to what extent these factors may have vitiated his result, his deduction is not conclusive.

A final comment must be made on cannibalism in mice. Topley's main conclusion is that there occurs a preepidemic rise of potential which gives rise to the epidemic wave. This may be excited by overcrowding, and he records that cannibalism is of very much greater incidence in overcrowded cages. Cannibalism was countered in our experiments by preventing overcrowding and by frequent inspection of cages. However, when it did occur, and twice on experimentally feeding the liver of an infected mouse to mice free of infection, a large crop of fatalities resulted. One cannot help thinking that many of the peaks in the epidemiological curves recorded by Topley *et alii* may be due to the fact that five to ten days before the peak there occurred a death due to the organism with which he was experimenting, and that the sign which on his curve in these places signifies a mouse eaten gives the cause of the subsequent peak [for example, reference (6)]. His evidence for a preepidemic rise of potential would be very much more convincing if this possibility could be eliminated.

Taking into consideration these factors, steps were taken to control the epidemic. Cages were designed with a separate sleeping compartment into which "Hygienic Flock", that is, flock made from the cuttings of new cloth, was put. A feeding compartment adjacent to the sleeping compartment made it almost impossible that the mice foul the food. The remainder of the floor of the cage was formed of quarter-inch mesh wire netting, through which fæces passed readily but young could not be lost. The cages were raised in trays to allow the refuse to fall through the floor. The temperature of the room was kept regulated at 67° to 70° F. The effect on the mortality rate became at once obvious, and in a fortnight the death rate had fallen to the summer level with a minimal number of specific deaths.

Summary.

An epidemic of mouse typhoid is recorded which shows:

1. That in mice ill with *Bacillus typhi murium* infection a falling body temperature is often present before they become moribund.

2. That falling atmospheric temperature increases the susceptibility to fatal infection.

3. That the addition of new "susceptibles" by breeding is a potent factor in increasing the mortality rate in an epidemic.

4. An acute infective ulceration of the gastric and duodenal region of the mice is frequent and often goes on to chronic "peptic" ulcer similar to the human type, if the mouse survives.

5. Lesions of liver and other organs similar in appearance to those usually termed lymphoid were found in mice experimentally infected with *Bacillus typhi murium*.

6. Steps taken for controlling the epidemic are described.

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EARLY SURGEONS OF THE SYDNEY HOSPITAL: THOMAS HENRY FIASCHI.[1]

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ALTHOUGH my paper will deal mainly with the late Dr. Thomas Henry Fiaschi, I wish to call to your minds the names of some of the early surgeons of Sydney Hospital:

Charles Nathan (1845-1865).
Farquhar MacCrae (1845-1846).
D. M. McEwan (1847-1859).
Sir Alfred Roberts (1855-1870).
James Robertson (1860-1862).
Sir P. Sydney Jones (1862-1872).
Chas. McKay (1863-1873).
Ed. Bedford (1866-1873).
G. Fortescue (1871-1874).
C. K. Mackellar (1873-1876).
Sir Normand MacLaurin (1874-1882).
W. W. Spencer (1874-1876).
Geo. Marshall (1875-1883).
Cosby W. Morgan (1877).
J. C. Cox (1877-1879).
T. C. Morgan (1878-1882).
Harman J. Tarrant (1880-1888 and 1891-1894).
A. J. Brady (1883-1884).
Mark W. Traill (1883).
J. M. Creed (1884).
G. P. M. Woodward (1884-1889).
Craig Dixon (1885-1886 and 1888-1890).
P. Muskett (1885-1887).
M. J. O'Connor (1887-1893).
A. MacCormick (1889-1890 and 1893-1897).
W. H. Goode (1890-1902).
Henry A. Ellis (1891-1892).
Thos. Fiaschi (1894-1896 and 1898-1911).

Dr. Thomas Fiaschi was born in Italy on May 31, 1853, and died in Sydney on April 17, 1927. His father was Lodivico Fiaschi, Professor of Mathematics at the University of Florence, and was a peace-loving man wrapped up in his literary pursuits. His mother was Clarissa Fisher, who came of an English family in which there were ten sisters, all of whom lived to over eighty years, one reaching the age of ninety-six years. Thomas Fiaschi's mother was a cultured woman, who before her marriage gave private tuition to the children of Prince Corsini of Florence. Dr. Fiaschi's sister, Eliza Fiaschi, was a woman of much the same nature as her brother—broad-minded, optimistic and jolly, always enjoying the company of young people. She was a graduate in music and singing of Milan and Leipzig, and for many years had a large number of pupils, amongst them many English and Americans. Thomas Fiaschi could trace his family back to 1560, to the old Tuscan family of Fiaschi of Tizzana, a small property in the district of Pistola, outside Florence. He learned to speak English at an early age from his mother, but had tricks of speech which revealed his Italian ancestry. He was a graduate of the University of Pisa, holding the degrees M.D., Ch.M. Amongst his fellow students at Florence was Banti, of international repute. From casual remarks that he made to me, there is no doubt that Thomas Fiaschi was a high-spirited youth. He came to Australia by sailing ship and landed at Brisbane about the time of the discovery of the Palmer River goldfields, and proceeded to Cooktown. Later, on the advice of Sir Normand MacLaurin, he went to Richmond, New South Wales, and thence to Windsor, where he had a large general practice and rapidly became well known. Visits to the outlying districts were done on horseback. In 1878 he returned to Europe and did post-graduate work at the London Hospital, coming under the influence of men such as Hughlings Jackson and Jonathan Hutchinson. He often quoted the former's remark to his class: "Gentlemen, there is a piece of the human brain sticking out for you to look at", so Fiaschi became proficient in the use of the ophthalmoscope and gained a knowledge of the diseases of the eye, which he put to good use in his general practice at Windsor. He was appointed to the honorary medical staff of the Windsor Hospital in 1878, and it was there that he laid the foundation of his surgical practice.

In 1883 Thomas Fiaschi put up his plate at 39, Phillip Street, Sydney. In later years he practised in Macquarie Street. In 1885 he again visited London and did more post-graduate work, and on his return gave up obstetric work and confined himself to medicine and surgery.

In 1894 he was appointed honorary surgeon to Sydney Hospital and held this position, with the exception of the time he spent at the Abyssinian Campaign and the South African War, till he reached the hospital retiring age in 1911. Having to retire in the fullness of his physical and mental vigour was a great sorrow to him. He said to a close friend: "When a surgeon has to leave his hospital his life is finished."

Thomas Fiaschi rendered distinguished service in the military forces at home and abroad. His first commission in the Army Medical Corps, New South Wales, was on March 24, 1891. He became lieutenant-colonel on March 1, 1901, colonel on June 8, 1911, and brigadier-general on retirement. He was Principal Medical Officer of the Second Military District from November 29, 1911, to October 30, 1917.

He served in the South African War of 1899-1902, first as medical officer of the First Contingent, New South Wales Army Medical Corps, and was at many engagements and displayed the utmost courage. In February, 1900, whilst searching for wounded in the Boer trenches he received the surrender of Cronji's forces. He was mentioned in the dispatches in the *London Gazette* on February 8, 1901, and April 16, 1901, and was awarded the Distinguished Service Order and Queen's Medal with five clasps.

He served in the Great War, 1914 to 1918, as Officer Commanding Number 3 Australian General Hospital at Lemnos until invalided to England, seriously ill with beri-beri. On recovery he served with the Italian forces on the Trentino front.

[1] Read at a meeting of the Section of Medical Literature and History of the New South Wales Branch of the British Medical Association on October 12, 1933.

Thomas Fiaschi took part in many public activities, especially in the wine-growing industry and Agricultural Society. He made many contributions to medical literature, of which may be mentioned his description of sliding hernia, quoted in Binnie's "Surgery"; also a paper on reintegration of the absent middle third of the tibia, which described a remarkable series of operations on the tibia of a child, with excellent result, which I was privileged to witness.

Having very briefly outlined certain historical facts, I should like to say something about the man as I knew him at Sydney Hospital. Many knew him better than I, but none admired him more. As a junior resident medical officer at the Sydney Hospital in 1906, my first impression of him still lingers. Tall and handsome, with erect military figure, quick step, keen searching eyes, he looked every inch a man. As he entered the ward his personality seemed to pervade everyone and everything. There was an air of expectancy in the ward. The house surgeon was there waiting for him, the sister and nurses standing to attention. His ward discipline was perfect. With a quick "good morning" he commenced his rounds, doing all the dressings himself, asking pertinent questions meanwhile. Nothing escaped his notice, and nothing was allowed to interfere with his hospital work—private patients could wait. There was always an uncertainty as to the mood he would be in. Usually he was quiet, courteous and amused at the quaint sayings of patients. At other times he was impatient, unreasonable and bad tempered. Any failure to carry out his instructions in detail roused him to fury. However, his anger was soon spent and he never bore ill-will. He particularly disliked dull nurses, especially those who were afraid of him, and was at times scathing in his remarks to them. He liked a capable nurse who stood up to him. One day when he was much annoyed, the sister in the ward, probably from nervousness, laughed. He glared at her and said: "What are you laughing at?" To his surprise she replied: "At you, Sir." His anger ceased at once and he laughed and said: "I am a bad tempered man, isn't it?" He advised a nurse of different calibre to eat a whale, as fish was good for brains. However, he was most popular with the nursing staff, which had an intense admiration for his devotion to duty. How frequently he used to say: "Disease knows no holiday. Duty comes before pleasure."

He had an effective method of dealing with a talkative patient when he wished to make an examination or do a dressing. He would ask the sister to get a glass of water and then he would most courteously ask the patient to take a mouthful of water and not to swallow till he gave the word. The patient thought the procedure was part of the treatment.

He looked the part as he entered the operating theatre clad in white singlet and trousers, wearing rubber boots reaching to the knee. He practised the most rigid asepsis and was very fond of carbolic acid in the treatment of septic wounds. His bone surgery was his best work, in my opinion. He was a sound surgeon who never operated without due thought. He was not a brilliant operator. If he set out to do any particular operation described by another surgeon, he paid him the compliment of following his description in every detail, for example, in gastro-enterostomy.

He was most insistent on careful after-treatment. An amusing incident occurred in this connexion.

As was his custom after an abdominal operation, he said: "Nurse, you will give him the Murphy's", meaning Murphy's saline solution. Some days after the operation he said the patient might have a small quantity of potato added to his diet. The patient said: "I have already had some." Fiaschi was furious that his instructions as to diet had not been carried out. However, investigation proved that the nurse, new to the ward and not knowing Fiaschi, had thought he had referred to potatoes facetiously as the Murphys.

Dr. Fiaschi relied on the clinical signs and symptoms in connexion with duodenal ulcer. If, on opening the abdomen, he could not feel the expected induration of an ulcer, he was wont to say: "There are two classes of ulcers—the ulcers you can see and feel and the ulcers you cannot see and feel. Naturally the latter are the more dangerous. We shall do a pylorectomy and gastro-jejunostomy."

When using a tourniquet Thomas Fiaschi always instructed a nurse to tell him every fifteen minutes how long the tourniquet had been applied. He had a habit of saying to the anæsthetist: "You will kindly have him deep now"; and very shortly afterwards: "Now we shall have him light"; and so on. One day he was so disgusted with the efforts of the anæsthetist that he told him to sit up in the visitors' gallery and he gave the anæsthetic himself.

If a nurse dropped a basin in the theatre he would say: "She should not be a nurse, she should be feeding the ducks."

Towards the end of the afternoon in the operating theatre he often told us about some of his experiences.

I remember him looking up as he had just finished stitching up the skin and saying: "You meet some funny people. I had a nurse once nursing for me, and whenever I went to see the patient I found her writing. I did not take any notice, thinking she was writing to her sweetheart, but, by George, she was a novelist and published a book in which I recognized myself as the particular villain."

Although on his regular operating day he expected everything to be ready without question, at an emergency operation at night he would put up with anything, as it was an emergency. He responded to a call to the hospital immediately. How familiar was his voice saying: "I shall be there in three minutes"; and so he was. We could hear his footsteps reechoing along Macquarie Street as he hurried along. One day he asked me how long it would take to have the theatre ready for an emergency operation. I replied: "Ten minutes." He said: "That is no good. Where would you be if a man were admitted with a wound of the heart? I should be able to operate in an instant."

He had a very keen sense of humour.

One day a nurse told him that the wound was a "pussy". He said: "Who taught you English? There is no such word as 'pussy', it is purulent." As he left the ward he saw a cat passing and said to his house surgeon, Dr. Arthur Colvin: "There goes purulent, isn't it?"

He liked the instrument attendant, the late Frank Wright, who had been there for many years and who used to ring up the instrument maker in regard to instruments being repaired, and tell him off in no uncertain manner; and he could be heard speaking outside the theatre. I remember Fiaschi stopping for a moment and listening, full of admiration, and saying: "Listen to him, he is fine." He was a man after his own heart.

Thomas Fiaschi was the first Chairman of the Board of Medical Studies at Sydney Hospital and took an intense interest in the work of the students. He was always most kind and courteous to them. If one of the students asked him a question, he would listen almost deferentially and then reply in detail. Several students who had been neglecting their work were brought before him and we expected that he would be most severe, but quite the reverse. He spoke most kindly to them and pointed out the error of their ways and urged them to make the most of their opportunities.

He was very fond of animals. He was often to be seen driving along in his sulky with two small dogs, which he called "Cricoid" and "Thyroid".

He was a man of so many parts that it is impossible for anyone to do justice to the subject of my discourse tonight. He was a fine surgeon, great citizen, soldier and patriot.

When the medical history of Australia is written, Thomas Fiaschi will be given an honourable place amongst the surgeons and medical pioneers of the Commonwealth. He read widely in medical and general literature, and his culture was evident whenever he spoke. The Board of Directors of the Sydney Hospital paid a well deserved and graceful tribute to his memory and to his many years of noble service to that great institution by naming one of the operating theatres in the Travers Pavilion "The Thomas Fiaschi Theatre". I trust that each year, on the anniversary of his death, whichever surgeon is operating in that theatre will address a few remarks to those in attendance there, recalling to them Fiaschi's honoured life, so that his fame may be handed on for generations to come.

THE CONTROL OF DIPHTHERIA IN A RESIDENTIAL INSTITUTION.

By H. M. LATHROP MURRAY, L.R.C.P. & S. (Edinburgh), D.P.H. (England),

Assistant Medical Officer, Greswell Sanatorium, Mont Park, Victoria.

SOME years ago, the writer, while holding the position of assistant medical officer of health in one of the midland counties of England, was called upon to assist in a consultative capacity the medical officer of a residential institution in which a case of diphtheria had occurred. The institution in question was a school with a population of 140, of whom 120 were boys between the ages of fourteen and nineteen, the remainder being members of the school staff. The patient, a boy who had recently entered the school, had, of course, been isolated; he was treated on orthodox lines and in due course made an uneventful recovery.

Immediate steps were taken to obtain swabs from the throats of every person in the school; these swabs were rushed post haste to the pathologist at the local hospital for examination. Each member of the school was then submitted to the Schick test. Forty-eight hours later a preliminary reading of the results of the Schick tests was done, and as a result it was possible to divide the inhabitants of the school into two groups: those who reacted to the Schick test, fourteen in number, and the remainder who did not react. These two groups were kept separate from each other.

After the expiration of a further twenty-four hours the reading of the Schick tests was confirmed; and by this time the report on the throat swabs was available. It was now possible to subdivide the two groups mentioned above as follows:

Group I. Schick-positive, swab-negative (14).
Group IIA. Schick-negative, swab-positive (11).
Group IIB. Schick-negative, swab-negative (115).

The figures in parentheses indicate the number of persons in each group. It will be noted that no person was found to be both "Schick-positive" and "swab-positive", and as may be expected from this, no further cases of diphtheria occurred in the next few days.

Unfortunately, on account of the expense which would have been involved, it was impossible to have virulence tests carried out. Hence it was necessary to assume that all those persons from whose swabs morphologically typical *Corynæbacteria diphtheriæ* were obtained were carriers. These, who, of course, comprised the members of Group IIA above, were segregated from the rest of the school; no naso-pharyngeal condition calling for surgical interference could be discovered in any of them, and so they remained segregated, living a monotonous life of energetic gargling at regular intervals until "negative" swabs were obtained.

The members of Groups I and IIB were immediately released from surveillance and were able to resume their normal school routine. On general principles I advised the immediate immunization of all the members of Group I. Some delay ensued before it was possible to obtain permission to carry out this procedure; but when this had been arranged, each member of the group was given three injections of toxin-antitoxin mixture at intervals of six days. Three months later these were tested again by the Schick test, and all now failed to react.

It is interesting to note that at the first test 13 boys out of 120, or 11%, gave reactions to the

Schick test, whereas out of the 20 adult members of the school staff only one reacted. This, of course, is to be expected on theoretical grounds, for the chances of natural immunization having occurred as a result of a subclinical or unrecognized infection are much greater in an adult than an adolescent.

The procedure which I have outlined is applicable to any community in which a case of diphtheria occurs. It has the great advantage of separating at the earliest possible moment the "Schick-positive" or susceptible group from the "Schick-negative" group, which will contain all the carriers of the community. The further subdivision of the "Schick-negative" group separates the carriers from those who, being "swab-negative", are harmless; while, should any persons be found who are both "Schick-positive" and "swab-positive", these should be kept under observation, as they are probably incubating diphtheria; they should be given antitoxin at the appearance of any sign of clinical diphtheria. It is desirable to have virulence tests carried out on the organisms from the swabs of all suspected carriers; but this means that there is a considerable delay while the progress of the inoculated guinea-pigs is observed. The additional expense of this procedure must also be taken into account.

Reports of Cases.

UNUSUAL AUTOPSY FINDINGS IN SUBACUTE BACTERIAL ENDOCARDITIS.

By A. R. BUCHANAN, M.D. (Melbourne), M.R.C.P. (London),
Medical Clinical Assistant, Alfred Hospital, Melbourne.

THE autopsy findings in the following two cases of subacute bacterial endocarditis are so unusual that, even in face of the voluminous literature on the subject, they seem worthy of record.

Case I.

E.D., a female, aged eighteen years, was admitted to hospital on March 16, 1932. She stated that she had been confined to bed for fifteen months, her actual illness commencing with an attack of pleurisy which lasted a fortnight. This was followed nine weeks later by an illness which lasted for five weeks, during which time the outstanding symptoms were swollen, painful joints, high temperature, dyspnœa on slight exertion, and continuous pain under the sternum. She stated that she had recently lost her appetite and had been sweating a great deal.

Examination revealed a pale, thin girl with a sallow, almost *café-au-lait* tint in the skin. The heart was enlarged, the apex beat being situated ten centimetres (four inches) from the mid-line in the fifth intercostal space. A loud systolic bruit was audible over the whole of the precordial region, its maximum intensity being heard over the third and fourth intercostal spaces on the left side, close to the sternum. The cardiac rhythm was rapid but regular. The liver and the spleen were not palpable on abdominal examination. There was moderate curving of the finger nails and early clubbing of the fingers. The Wassermann test gave no reaction. The urine contained albumin, and on microscopical examination many red blood corpuscles were found.

Her subsequent course in hospital was slowly progressive, with intervals of remission and with occasional exacerbations of fever. No change was noted in the heart sounds, but during June, 1932, the spleen enlarged and became palpable and remained so until death. Early in July diarrhœa became troublesome and persisted, despite appropriate treatment. She died on July 30, 1932.

Post Mortem Findings.

Necropsy was performed on August 1, 1932. The salient features were as follows.

Heart.—There was some enlargement of the heart, due mainly to hypertrophy of the right side. The pulmonary and tricuspid valves were extensively destroyed, and the remnants bore large ragged masses of soft vegetations. There was a small perforation of the *pars membranacea septi* with soft ragged vegetations on its right side. The aortic and mitral valves were normal. The pulmonary artery appeared to be smaller than normal. The picture presented was that of infective endocarditis supervening on a congenital malformation, namely, a patent interventricular septum with probable stenosis of the pulmonary artery. The proof of the latter was rendered doubtful by the extensive destruction of the pulmonary valves.

Lungs.—Scattered infarcts were present throughout both lungs, one of these showing signs of breaking down to abscess formation.

Spleen.—The spleen was found to be enlarged to four times the normal size. The Malpighian corpuscles were prominent and extensive amyloid infiltration of the "sago" type was present throughout the organ.

Liver.—The liver was slightly enlarged and showed extensive amyloid infiltration.

Kidneys.—The kidneys were very pale, the subcapsular surface very smooth and mottled, and light amyloid infiltration was present, particularly in the cortical zone.

Microscopic Features.—Microscopic sections of the liver, spleen and kidneys confirmed the presence of amyloid disease. The other organs showed no apparent pathological change. No other cause for the amyloid disease was detected, even after a careful and intensive search.

Case II.

C.C., a male, aged sixteen years, was admitted to hospital on June 16, 1932.

On admission he stated that he had been quite well till eight weeks previously, when he had suffered a severe epistaxis. From this he recovered and felt quite well until six days before admission, when he had another severe epistaxis.

There was nothing definite in his past history and no indication of previous rheumatism, but his mother stated that she had been told some years before that he had a "rheumatic heart". On examination he was exceedingly pale, with a faint icteric tint in the skin and conjunctivæ. The heart was enlarged, the apex beat being 11·25 centimetres (four and a half inches) from the mid-line in the sixth intercostal space. There was a marked systolic bruit, audible at the apex, which was conducted to the axilla; there was also a faint apical diastolic bruit.

At the aortic base there was a well marked to-and-fro murmur. The cardiac rhythm was rapid but regular. The abdomen was enlarged and there was a large splenic tumour filling the left loin and extending down almost to the pelvis and across the middle line.

The urine contained a trace of albumin, and red blood corpuscles were present in the deposit.

A blood examination revealed the presence of marked secondary anæmia. The erythrocytes numbered 2,160,000 per cubic millimetre, the leucocytes numbered 5,100 per cubic millimetre, the hæmoglobin value was 20%, and the colour index was 0·46. The film showed some polychromasia only. The differential white cell count was normal. The bleeding time was normal. The coagulation time was slightly prolonged. The clot retraction was normal.

Attempts at blood culture were made on a number of occasions and were repeatedly without result.

Whilst under observation the patient became progressively worse, running a continuous hectic temperature; death occurred on August 17, 1932.

Post Mortem Findings.

Necropsy was performed on August 18, 1932. The salient features were as follows: The heart was found to be enlarged, largely because of hypertrophy of the left ventricle. The aortic and mitral cusps were extensively destroyed and the remnants bore large masses of exuberant soft friable vegetations.

The kidneys were enlarged and congested, each weighing 212·6 grammes (seven and a half ounces). The surface of each was smooth, with many subcapsular petechial hæmorrhages of the flea-bitten type. On section, areas of focal nephritis were apparent, and there were a number of anæmic infarcts in each kidney.

The spleen was enormously enlarged, its dimensions being 30·5 by 19·6 by 8·25 centimetres, and its weight 1,450 grammes (52 ounces). A large anæmic infarct was found to occupy the central fifth of the organ. The remainder of the spleen showed no gross macroscopic change. Microscopic section of the spleen showed marked congestion and hyperplasia of the splenic pulp, but no suggestion of the characteristic changes of other types of gross splenomegaly was found. The other organs showed nothing noteworthy.

Summary.

1. Two cases of subacute bacterial endocarditis with unusual *post mortem* findings are here recorded.
2. The first case is remarkable because amyloid disease apparently resulted from an infective endocarditis. A survey of the literature reveals no record of the occurrence of such a complication in this disease. Amyloid disease has been described by several authors as a rare complication of chronic rheumatic processes, but in this case there is no evidence in the history to suggest a previous rheumatic infection, and the autopsy findings point to the fact that the cardiac lesion was ingrafted on a congenital defect.
3. The second case is noteworthy for the enormous size of the spleen, which, as far as can be ascertained, is a most unusual occurrence in this disease.

PLUMBISM CAUSED BY THE INGESTION OF LEAD PAINT.

By H. S. HARPER, M.B., Ch.M. (Sydney),
Cooran, Queensland.

IN view of the increasing interest now being taken in the various causes of plumbism in children, its devastating effects manifested in later years, and the fact that but few cases are reported from country districts, the following case will be of interest to and may help the cause of those who advocate the abolition of lead-containing paints.

P.O'R., a female, aged four years, was brought to me on July 18, her parents stating that during the preceding fortnight the child had fallen several times, appeared to want to lag behind while out walking, and had developed a peculiar gait. She had vomited on several occasions, but, apart from being cross for the past month, had appeared to be otherwise healthy.

On examination the patient was found to be a well nourished child. The skin of the neck, body and limbs was pale, but the cheeks were flushed and the lips a good colour. The temperature and pulse rate were normal.

The patient had the typical foot-drop and high steppage gait of a person with bilateral external popliteal nerve paralysis.

Nothing abnormal was found in the heart, chest or abdomen. The tonsils and adenoids were hypertrophied. The urine contained no sugar or albumin.

On making further inquiries from the parents, I learned that the child had a habit of sucking its thumb and that the paintwork on the veranda where she usually played was dry and powdery.

A provisional diagnosis of lead poisoning was made and the patient was then seen by Dr. L. J. Jarvis Nye, of Brisbane, who agreed with the diagnosis and supplied the following details of blood examinations:

Erythrocytes, per cubic millimetre ..	3,620,000
Halometer	4·6
Colour index	1·1
Hæmoglobin	80%
Leucocytes, per cubic millimetre	18,000
Polymorphonuclear cells	35%
Lymphocytes	32%
Eosinophile cells	30%
Transitional cells	2%
Basophile cells	1%
Stippled cells	62 per 100 white cells

An occasional poikilocyte was seen. The Klein test gave no reaction.

A twenty-four hour specimen of urine did not reveal the presence of any lead.

A sample of the powdery paint from the veranda posts was examined and gave a positive result to a test for lead.

No other source from which the child might have obtained lead could be discovered, making it fairly obvious that the powdery condition of the lead-containing paint and the thumb-sucking habit were the two factors concerned in causing plumbism in this patient.

The patient is being treated by daily doses of magnesium sulphate, together with a mixture containing potassium iodide, sodium citrate and sodium bicarbonate. Plaster of Paris splints were made for the legs and feet. After five weeks' treatment the patient is gradually regaining power in the extensors of the feet.

An interesting feature of this case is that about twelve months ago the child was brought to me with a history of having fallen from a height of about three feet on to her left arm. At that time I noticed that she had wrist-drop; the forearm, especially close to the wrist, was tender, but there was no definite evidence of fracture or sprain. The forearm and hand were splinted for a fortnight, and on removal of the splints appeared to be quite normal in every respect. The mother now informs me that one month later the child developed wrist-drop again without having had a fall. This passed off in a few days.

Acknowledgement.

I am indebted to Dr. L. J. Jarvis Nye for his full reports on the blood examinations *et cetera*, and for the keen interest he has taken in the case.

Reviews.

LIGHT THERAPY.

IN the absence of any teaching authority, very many, and possibly the majority, of those who are practising actinotherapy in Australia have been compelled to work out their own technique. Until this hiatus in medical instruction is filled, either by making the science of physiotherapy a necessary part of the medical curriculum or by post-graduate instruction, such a manual as "Actinotherapy Technique", published and issued by the Sollux Publishing Company, will be of considerable value.[1] Both users and those who contemplate the use of the ultra-violet and infra-red rays will find this work of considerable assistance. It is a summary of treatment based on many books and papers dealing with actinotherapy, a term which, while it is usually applied to ultra-violet treatment, also includes in this book the use of the infra-red rays. Every suggested method mentioned is referred to the medical man who has advocated it and to the book or journal in which his views were expressed. The book is in this way a synopsis of many opinions and an attempt to standardize treatment.

Chapters dealing with physical data, physiological effects, methods of administration, and dosage are included. In a foreword, Sir Henry Gauvain writes: "With the aid of this book the practitioner, with the requisite equip-

[1] "Actinotherapy Technique: An Outline of Indications and Methods for the use of Modern Light Therapy", with foreword by Sir Henry Gauvain, M.D., M.Chir., F.R.C.S.; 1933. Slough: The Sollux Publishing Company; Sydney: W. Watson and Sons, Limited. Crown 8vo., pp. 134. Price: 6s. net.

ment, has at hand, in convenient form, an aid to special treatment which is practical, illuminating and helpful." In the introduction the compilers point out that most ailments call for due use of the triad which comprises physical therapy, medicine and surgery. "Actinotherapy Technique" includes only a part of one of these, but while mention is made only here and there of surgical or medical treatment, there is no attempt in the book to exclude other methods which may be combined with actinotherapy. With this attitude no one can find fault.

CLINICAL HÆMATOLOGY.

ANYTHING that Price-Jones has to say about hæmatology merits attention, and the third edition of his "Blood Pictures: An Introduction to Clinical Hæmatology",[1] is of interest not only intrinsically, but as a reflex of a personality. The author has packed into a small book of under eighty pages an astonishing amount of information on interpretation of blood counts. That is all the book pretends to be, and that is all it is. It sets out all the conditions in which a blood picture can be of definite value in diagnosis, and as these are limited in number, the book is small—72 pages. The interpretations given of typical counts are reasonable and not marred by that over-enthusiasm which is so often misleading. This book, by an acknowledged expert, can be recommended to those seeking a sound interpretation of blood pictures and to those seeking to know when hæmatology can be of definite help. There are coloured plates of unimpeachable accuracy, and it would have been of help to the amateur to let him know whether they are all drawn to the same scale, since a scientific outlook can be based only on proper standards. Price-Jones's remark on page 28, that the limits (of the red cell count) for all healthy persons are roughly 4,000,000 to 6,400,000 per cubic millimetre and that counts outside these limits should be regarded as not healthy, is not only interesting, it is highly significant, if taken in conjunction with the well merited emphasis now being laid on the secondary anæmias and their relation to malnutrition. We must, of course, refer to the Price-Jones curve, a very wonderful, though tedious work. Price-Jones insists that here it is the anisocytosis or variability in size that is significant in pernicious anæmia; "this sign", he says, "is even more a constant feature than the increased mean diameter of the red cells in this disease".

Within its limitations this is an excellent little book.

MATERIA MEDICA AND THERAPEUTICS.

THE thirteenth edition of "A Treatise on Materia Medica and Therapeutics", by R. Ghosh is to hand.[2] This is an excellent work and the present edition maintains the high standard of previous issues. A drastic revision has been made in accordance with the British Pharmacopœia, 1932. Most of the drugs deleted from the pharmacopœia have been omitted, but some are still retained as being of therapeutic value or having important pharmacological actions. The classification of drugs has been modified and, instead of the section dealing with general pharmacology, the action of drugs, with their physiological bearing, is discussed before each main group. The book is smaller than previous editions. The recommendations of the pharmacopœia as regards prescriptions have been indicated, but not followed. A special section deals with indigenous Indian drugs which are of interest to Australia, as several of the plants mentioned are indigenous also to Australia. Of "Salyrgan" it is stated that it gives better results as a diuretic than "Novasurol" and does not, like the latter, cause diarrhœa. Another complex mercury compound, "Metaphen", is used for sterilizing instruments and the skin. It does not injure instruments or precipitate proteins. As regards the gold treatment of pulmonary tuberculosis, "Solganal" and "Solganal-B" and "Sanocrysin" (*sodii aurothiosulphas*) give good results in early cases, but results are not so favourable in advanced cases. In the treatment of syphilis "Bismarsen" (bismuth arsphenamine sulphonate) is mentioned as containing 13% of arsenic and 24% of bismuth. "Fuadin" is a trivalent antimony compound with pyrocatechin sodium disulphonate for bilharziasis. Complex iodine compounds for diagnostic purposes are well described, such as "Abrodil" and "Uroselectan" for the urinary tract, and lipiodol for the bronchial tree, pulmonary cavities and spinal cord. A fatality due to lipiodol is mentioned, but it is more likely that such fatality was due to the preliminary application of cocaine. "Avertin" is stated to have caused several deaths. It is pointed out that a dose sufficient to induce anæsthesia may cause failure of respiration, and "Avertin" is therefore used in smaller doses as a basal narcotic supplemented by light administration of ether or a local anæsthetic. Amylene hydrate, "Nirvanol", "Theominal" and "Pernocton" are all adequately treated. But "Nembutal", on page 296, is wrongly stated to be phenobarbital sodium; on page 299 it is said to be sodium ethyl butyl barbituric acid, which is not quite correct. Suprarenal extract for Addison's disease, desiccated stomach for pernicious anæmia, and "Optochin" for pneumococcal infections, all receive due consideration, the last named being unsafe in human beings on account of its effect on the eye. Choline, acetyl choline and halibut liver oil (with vitamins *A* and *D*) bring the book thoroughly up to date. Of the Calmette and Guérin vaccine for tuberculosis prophylaxis it is said not to be strong enough to prevent infection, but that it prevents the infection from becoming malignant and renders it benign. The section on ergot and its constituents is particularly clear and lucid. Anaphylaxis and allergy, vaccines and serums, non-specific protein therapy and organotherapy are very well expounded. "Atebrin" and "Plasmoquine" in malarial infections also are considered. The part dealing with vitamins is not complete. No mention is made of carotene or of hexuronic (ascorbic) acid in connexion with vitamins *A* and *C*. The pellagra-preventive vitamin is called B_2 or *F*, which is wrong; it should be *G*. On page 564 bulbous lesions are said to follow boric acid administration. This should obviously be bullous. In spite of a few defects, this work continues to be of the highest excellence.

REJUVENATION.

SERGE VORONOFF, one of the most famous of the modern protagonists of the method of glandular grafting in the search for the elixir of youth, has, in defence of his thesis, published a small book on the springs of life. This is not a detailed scientific treatise; it is rather a popular exposition of his ideas, with proofs drawn from his work on the lower animals and man.[1] It is worthy of note that the author advocates multiple grafting, and although his preliminary remarks do not suggest that he places the pituitary and thyreoid glands on the same level of functional importance as the gonads in this regard, he appears to utilize them as well. In speaking of the criterion of age, he asserts that: "*Le but de la vie n'est autre que l'amour.*" Perhaps the case history he relates of a lady who hastened to be grafted because her husband had left her on account of her age, but who, after grafting, did not waste her regained youth on him, saying "*il ne le mérite pas*", illustrates this. The result here must have been very good, for the husband came also to undergo the rejuvenating procedure himself. It will be gathered that there are things in this little book that are unconsciously amusing; this is particularly so in the descriptions of the personalities of some of the apes that the author has used for experimental purposes.

A seeker for a physiological treatise on the subject will not find it in "*Les Sources de la Vie*", but anyone who wishes to glean some information of the ideas and general methods of Voronoff will find it clear and interesting.

[1] "Blood Pictures: An Introduction to Clinical Hæmatology", by C. Price-Jones, M.B.; Third Edition; 1933. Bristol: John Wright and Sons, Limited. Demy 8vo., pp. 72, with illustrations. Price: 6s. 6d. net.

[2] "A Treatise on Materia Medica and Therapeutics", by the late R. Ghosh; Thirteenth Edition by B. N. Ghosh, F.R.F.P. & S.; 1933. Calcutta: Hilton and Company. Crown 8vo., pp. 727, with illustrations. Price: 12s. 6d. net.

[1] "Les Sources de la Vie", by Serge Voronoff; 1933. Paris: Fasquelle Editeurs. Demy 8vo., pp. 184. Price: 15 francs.

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THOUGHTS, WORDS AND WORKS.

WHEN man in his evolutionary development learned to express thoughts in words, he established his supremacy over the rest of the animal kingdom. Man obviously had thoughts before he learned to express them. Possibly animals also have thoughts. We do not know whether they have, but at least we may say that he is on debatable ground who would maintain that they have none. It has been held that animals have a simple consciousness in which the knower, the knowledge and the thing known are undifferentiated; that they have no self-consciousness. That animals have consciousness is obvious; moreover, they are able to communicate certain emotions to their companions. The consciousness of an infant is like that of an animal. It is when a child becomes conscious of self that his power of thought as we know it begins to manifest itself. Heredity and environment both play their part and with the growth of the cerebral cortex the child, from being a creature without self-consciousness, gradually develops—he is educated or drawn out and becomes a reasoning being. According as the mind is made receptive to external impressions and is kept in a receptive state, so will the intelligence develop. Intelligence reaches its highest peak by the creative power of thought. The potentialities of human thought are described by Edward Carpenter in his book, "The Art of Creation", when he writes that creation is "a process . . . which we can see at any time going on within our minds and bodies, by which forms are continually being generated from feeling and desire; and gradually acquiring more and more definition, pass outward from the subtle and invisible into the concrete and tangible . . . . It is the foundation of all human art." It is interesting in this connexion to recall a mental state in which man is able to stop thinking, to have his mind a blank. This is a state cultivated by the Yogi in India; it is a particularly receptive condition by the assumption of which the devotee prepares himself for that contemplation peculiar to those of his kind. This is mentioned merely in passing, for to discuss it would lead into the subject of states of consciousness higher than those experienced by the average man. In their well known book, "The Matrix of the Mind", Wood Jones and Porteus point out that thought is almost always a motor response, that is, it is almost invariably subvocalized or vocalized. Man thinks mostly in words, and though it may be possible for man to think in images, few people, if any, do. Thus thoughts and words are inseparably bound together, and thought is the life of man.

As is man's thought, so will be his creative power, his initiative and his capacity for appreciation. Thoughts are expressed by words and by actions or behaviour. An intelligent man, therefore, should for his own sake so order his speech and his comings and goings that they may express the best that is in him—he would not allow his creative genius to give out an unworthy product. By this expression of the best he is continually educating himself, and he finds ever and anon, paradoxical though it may sound, that the best that is in him is not the best of which he is capable. Receptivity and open-mindedness—a refusal to accept as final any of his favourite dogmas, however dear to him they may be—are the factors that make continual development possible. The man whose mind runs in one

groove displays that groove at every turn—the business man with his morning paper, his unceasing talk of finance and his constant care for markets and prices; the type of medical practitioner whose "cases" shut in his whole field of mental vision and circumscribe his actions; the politician with his vote-catching expedients dominating his attitude to men and affairs; and countless types to be found in every walk of life. As with works, so with words. There are words of wisdom and words of foolishness, words of sense and words of nonsense. At one end of the scale we have the words of a Solomon or a Plato, of a Christ or a Buddha, of a Francis Thompson or a Whitman. At the other end we have the drivelling nonsense of a dement. The words of most men do not reveal much depth of thought, sometimes because their thoughts have no depth, sometimes because they do not wish to reveal themselves, sometimes because they are too lazy. For all that, a man's words may generally be taken as reflecting his thoughts.

Since words and works are but the expression of thought, and since self-education is possible throughout life, it behoves each man, if he would rise to greater heights, to eschew words and works unworthy of him. The intelligent man differs from the fool in that his pleasures have a wider range and a greater variety. But he knows that pleasures of the senses, if controlled and enriched by the intellect, are most satisfying and most lasting; and his sense of values makes him choose things that the fool would disregard. This is why it takes a wise man to make a perfect fool. "The Preacher" in the book of "Ecclesiastes" found that "all was vanity and vexation of spirit, and there was no profit under the sun". But he also said that "the wise man's eyes are in his head; but the fool walketh in darkness".

Current Comment.

SCARLATINAL NEPHRITIS.

SCARLET FEVER is not nearly so virulent a disease as it was in the earlier years of this century; it would seem also that it is not so frequently complicated by nephritis. Still, scarlatinal nephritis occurs in some 5% of cases, and as the incidence of scarlet fever in the community is high, the importance of nephritis as a complication is manifest. Presumably the changes in the kidney are due to the action of a toxin; but why the toxin has these effects in only a certain small percentage of cases is a matter for conjecture. A. A. Osman, H. G. Close and H. Carter discuss the possible ætiological factors in a recent paper based on work carried out by them in the years 1927 to 1929.[1] They rightly limit the term scarlatinal nephritis to the acute, diffuse, glomerular or glomerulo-tubular nephritis that occurs generally between the fifteenth and thirty-fifth days and most frequently about the twentieth to the twenty-second day of the illness; they do not apply the term to the early febrile albuminuria, "the rare cases of acute interstitial nephritis, or the various forms of focal nephritis which may be met with at other stages of the disease". The main object of the investigation was to determine the difference between scarlet fever complicated by nephritis and scarlet fever that was not so complicated. A study of the acid-base balance throughout the course of the disease suggested itself for various reasons, chief among them being the finding by Carter and Osman that large doses of alkalis appeared to render scarlet fever patients less liable to nephritis. It was noted, also, that Colwill had found that nephritis and febrile albuminuria were more apt to occur if the urine was persistently acid and that in some cases there was a tendency for the urine to become more acid between the tenth and thirtieth days of the illness.

As they had no facilities for the direct measurement of the hydrogen-ion content of the blood by a reliable method, Osman, Close and Carter investigated the alkali reserve of the blood, measuring the serum bicarbonate content by the method of Van Slyke, Stillman and Cullen. They examined 28 patients suffering from scarlet fever of average severity, and found that in the majority of cases there was a diminution in the serum bicarbonate content during the first four or five days of the disease, followed by an increase, often to above normal, reaching its maximum about the fifteenth day, then gradually falling to normal during the next twenty or thirty days. In other words, there is an initial acidosis followed, in many cases, by an alkalosis, which slowly subsides during the later stages of the disease. It is seen that the serum bicarbonate content is about normal or above during the period in which nephritis is most likely to occur—between the fifteenth and thirtieth days. In a number of cases (seven of twenty-eight in which these investigations were made), however, there is a practically unvaried acidosis throughout, or the serum bicarbonate content rises not quite to normal, or having risen to normal, falls rapidly to below normal again. In this group there is an acidosis during the period in which nephritis is most likely to occur. Osman, Close and Carter found that the administration of alkalis (they do not state what alkalis) by mouth in a dose of

[1] *Guy's Hospital Reports*, July, 1933.

thirteen grammes a day prevented acidosis. In a group of seven cases, 150 cubic centimetres of glucose a day were administered to each patient without any apparent effect on the serum bicarbonate content, even in the early period of the disease, when ketone bodies could be found in the urine. During the secondary acidosis there is no ketonuria, and it is quite certain that this condition is not due to ketosis. As the primary acidosis was unaffected by the administration of glucose, the suggestion is that it is not caused by ketosis either.

Osman, Close and Carter also investigated a number of patients suffering from acute tonsillitis, a number suffering from diphtheria, and a number suffering from measles. Roughly, the same changes were found in tonsillitis as in scarlet fever; but observations were limited, as the patients were not admitted to hospital. The patients suffering from measles had a primary acidosis but apparently no secondary acidosis; these patients were discharged on the twenty-first day of the illness and were not examined afterwards. The patients suffering from diphtheria had no acidosis at any stage of the illness, although they had ketonuria at the onset. Osman, Close and Carter remark that the secondary acidosis of scarlet fever and of acute tonsillitis is probably in some way related to the nephritis that may follow these diseases. They point out that secondary acidosis does not occur in diphtheria, a disease that is seldom followed by nephritis. They have also noted that persons suffering from scarlet fever appeared to be rendered less liable to nephritis by the taking of alkalis. Osman and Close showed in 1930 that the serum bicarbonate content of old people was normally above the average for the healthy of both sexes; it is therefore of interest to note that old people are seldom affected with acute glomerular nephritis, and when they are, the disease is comparatively mild. Nephritis occurred in only one case investigated by Osman, Close and Carter; for some days before the renal lesion became obvious the urine was unduly acid and there was no alkaline tide. Summing up the evidence, they suggest that the secondary acidosis in scarlet fever is "a manifestation of a state of hypersensitivity which is an important, if not essential, factor in the ætiology of scarlatinal nephritis". Lukens and Longcope injected killed hæmolytic streptococci into the renal arteries of rabbits, some of which had been previously sensitized by the intracutaneous injection of living organisms of the same strain; far greater numbers of the sensitized than the non-sensitized developed glomerulitis. It is further pointed out that acidosis is known to occur in some allergic conditions. On the other hand, there is a leucopenia in allergy, a leucocytosis in the acidosis of scarlet fever; also the blood pressure tends to fall in anaphylaxis, and the temperature is generally below normal.

Of course, Osman, Close and Carter do not pretend that they have solved the problem of the causation of scarlatinal nephritis; they merely offer a suggestion for consideration. They write: "Sufficient direct evidence however is lacking and we must be content merely to record the changes found." Apart from the directions it gives for future research, the paper has its chief value in the possibilities suggested in the way of prophylaxis against scarlatinal nephritis. Obviously there is much more to be learnt; for example, if the secondary acidosis is due to hypersensitivity and the hypersensitivity is the cause of the nephritis, how can correction of the acidosis alone prevent nephritis, as it seems to do in many cases?

THE ANGINA OF EFFORT.

SINCE further knowledge has been gained of the relation of the more severe forms of cardiac pain to coronary vascular catastrophes the field of angina has been considerably narrowed. Perhaps a good deal of the controversy that arose over the cause of the pain has now been stilled by a recognition of the various pathological conditions underlying the anginal attacks. Undoubtedly in cases of coronary occlusion the pain arises from the heart. It would seem probable that the same origin holds good for some of the less serious types of cardiac pain, and in support of this may be cited some recent work on the subject.

E. J. Wayne and L. B. Laplace have made a study of eleven patients in whom effort, and effort alone, induced anginal attacks.[1] They have thus dealt with a selected and delimited type of angina and, furthermore, one that can be experimentally controlled, for it arises solely from the stimulus of exertion. The plan followed was to ask the patient to walk up two steps to a platform, cross, descend on the other side, and then turn and repeat the manœuvre until pain was felt in the chest. Each patient selected his own rate of walking, and this rate was then used for each test. As soon as pain was felt the patient was asked to sit down, and pulse rate, respiration rate and blood pressure readings were taken, previous observations having established the resting levels. The number of ascents to the platform was noted; also the time taken for the pain to disappear. Fifteen minutes' rest was allowed before a test was repeated. The possibility of suggestion influencing the results was disposed of by trying the effect of insinuating that a drink of water or saline solution would improve the patient's performances; any persons responding to such suggestion were rejected as unsuitable. In this way constant observations could be made and the principle of exact measurement was applied to the experiments. As regards the propriety of inducing anginal attacks in these patients, it must be pointed out that no unusual exertion was called for, and the test merely duplicated under standard conditions almost daily happenings in their lives.

Over four hundred attacks of effort angina have been studied in this way. The pain was nearly always retrosternal in position; in one case only it started below one shoulder blade. It was never throbbing or discontinuous, and seemed to possess

[1] *Clinical Science Incorporating Heart*, Volume I, Number I, 1933.

a distinctive quality that the patients described as "burning" or "gripping". Neither anxiety nor pallor was induced by the degree of pain experienced in the tests. Observations of the pulse rate and blood pressure showed that the appearance and disappearance of pain were not related to the blood pressure, but nearly always were directly related to the heart rate. Thus in a few instances the patient was allowed to perform the test exercises immediately after the pain had disappeared. The pulse rate at this time had fallen to its original level, though the blood pressure was still raised. The pain that had disappeared, despite the elevated blood pressure, now returned as the pulse rate rose. The effect of various drugs was tried on these patients also. Atropine caused a diminution in the amount of exercise required to produce pain and a prolongation of the attack. Amyl nitrite reduced the duration period of the pain, but no blood pressure fall was noted when the pain disappeared. Nitroglycerine increased the tolerance to exercise in the majority of cases, though at the moment when pain was complained of, the blood pressure was not raised above its normal for the patient. Erythrol tetranitrate, in spite of its favourable reputation in the treatment of angina, caused alterations in blood pressure and pulse rate, but increased the tolerance to exercise only in three cases. Euphyllin was tried in a few patients who responded well to nitroglycerine, but only a slight increase in exercise tolerance was gained. Prolonged rest in bed had a favourable effect, but chiefly in patients with a short history of anginal attacks. Pressure on the carotid sinus produced a definite diminution in the duration of the pain.

The bearing of these results upon the cause and site of the pain is interesting. Rise in the aortic blood pressure does not seem to be a factor in the genesis of pain in these cases. The rise in the heart rate seems to be of much greater importance, and as this raises the amount of energy actually expended by the heart without increasing the amount of the coronary blood flow, the pain would appear to be related to myocardial ischæmia. The relief afforded by the nitrites is due to a dilatation of the coronary vessels and not to a fall in systemic blood pressure. Other work has recently shown that the effect of the nitrites on general blood pressure is very variable and unreliable, which again centres our attention on the circulation in the heart itself rather than in the body as a whole.

It must not be thought that such studies as this dispose of the occurrence of pain of true aortic origin. Perusal of the case histories, particularly of those in which subsequent autopsy was performed, shows that the lesion, where demonstrable, was coronary. In only a few of the cases was disease of the aorta definitely detected, and thus there still remains that not inconsiderable group described by Allbutt in which there is sclerosis of the ascending limb of the aorta. If corresponding studies could be made of this group they would no doubt be of equal interest and value.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Moonta on September 30, 1933, DR. E. BRITTEN JONES, the President, in the chair.

Appendicitis and Its Treatment.

DR. H. R. POMROY read a paper entitled: "Appendicitis and Its Treatment" (see page 867).

DR. I. B. JOSE congratulated Dr. Pomroy on the admirable manner in which he had put the case for the more conservative treatment of certain cases of acute appendicitis and on the clear description of the pathology on which the theory of "delayed operation" was based.

He agreed that there was a definite indication for the delayed method, so long as it was applied with judgement rather than over-enthusiasm, and always with strict adherence to the rigid *régime* of rest to the bowel and constant supervision. Otherwise, to teach its adoption in so wide a manner would be definitely dangerous. He did not consider that Dr. Pomroy's figures of the increasing death rate per million were necessarily due to the almost universal plan of immediate operation, but rather to the increasing incidence of the disease, and though the mortality and morbidity of cases complicated by abscess or general peritonitis had not improved, the percentage mortality of all cases of acute appendicitis in the Adelaide Hospital statistics had steadily improved in each five-year period from 1905, owing mainly to earlier diagnosis and earlier admission to hospital.

Dr. Jose said that in the early stages, with the disease confined to the appendix, there could be no disagreement that immediate operation was the only course. In this period it was diagnosis that was all-important. Wilkie had frequently stressed the importance of recognizing the dangerous obstructive type, which often began with pain, frequently severe and umbilical in site, with a minimum of the signs of peritonitis. It was of extreme importance to teach the earliest symptoms of appendicitis in its various guises, but he considered it dangerous to rely at this stage on trying to discriminate the various types. It was essential and enough to diagnose appendicitis or not at once, for it was only operative treatment at this earliest stage that would reduce the mortality below the present 1%. He said that of the cases seen from about the third day onwards conditions differed, and some idea could be formed of the pathology present and the likely course, or an estimate could be made on which treatment must be based. Some cases were definitely resolving and were safe to leave for an "interval" operation.

Another group were those with widespread peritonitis—rigidity, tenderness, raised pulse rate and toxæmia—which definitely required immediate interference. Again, for some time the disease was still practically confined to the appendix with, perhaps, a few light adhesions and local peritonitis, but without a palpable mass. These appendices Dr. Jose still considered safest removed immediately, as many of the patients became rapidly worse on waiting at all, or else eventually developed an abscess and so greatly lengthened the morbidity period.

Then there were cases in which a palpable mass had formed—either a brawny mass with ill-defined margins (a similar type in the pelvis was felt *per rectum*) or at a later stage a more sharply defined mass which on exposure showed a lump, well localized by dense adhesions, containing a small abscess and little or no peritoneal reaction adjacent. To interfere with the natural protection in these instances was distinctly not indicated. Again, late cases with ileus developed should be left alone. And, finally, if in pelvic peritonitis there was doubt as to whether the appendix or Fallopian tube was the cause, it might be safer to await developments.

To discriminate all these types which greatly overlapped required considerable clinical judgement, and Dr. Jose considered that to advocate delayed operation too

indiscriminately after the early stages would surely increase the incidence of the complication of peritonitis, subphrenic abscess and pylephlebitis, as used to occur thirty years before.

DR. BRONTE SMEATON said that Dr. Pomroy had given them some very important facts and opinions to consider. Dr. Smeaton's memory went back to the time when an interval operation was the object of treatment, and he remembered many unforeseen fatalities during the period of waiting. It was difficult to express the intensity of the disease in terms of time; virulence of infection, position, previous attacks *et cetera* caused variations in the course, while the time factor remained constant. They had heard some most interesting statistics as to results of operation; these were largely from English hospitals and were most valuable; but they must remember that the technique of operation varied. The split muscle incision was almost universal in Australia, while an approach from the median side of the appendix was used a good deal in England. When one remembered that the localization of the disease was the intention of the phenomena accompanying it, and that the most vulnerable wall was on the median aspect of the infected area, where the distended stationary coils of small intestine formed the barrier, one saw how dangerous was a route that disturbed this defence. The best operation was that accompanied by the least dissemination of infection, and it was the search for this safety that prompted Dr. Pomroy's paper.

DR. J. RIDDELL appreciated the paper read by Dr. Pomroy. Reviewing the progress of the treatment of acute appendicitis during the past thirty years, he was surprised to learn that the mortality rate in the hospitals mentioned was as high as 7%. Though not in a position to quote statistics, he did not think the death rate in private practice would be so high. The patients were generally seen and operated on earlier, whereas a large number of patients sent to hospital had probably been under observation and medical treatment for a few days.

He agreed that the earlier acute appendicitis was operated on the better. Regarding delayed operation in cases seen thirty-six to seventy-two hours after onset, though this might be a safe procedure in general hospital practice, through lack of constant observation facilities by skilled house surgeons and judgement in private practice as to the exact time to step in, it might be fallacious and lead to disaster and the greater likelihood of secondary complications. He would favour immediate operation in all cases of acute appendicitis, unless obvious signs of abatement were in evidence. The danger appeared to be ofttimes in attempting too much, especially the removal of the inflamed appendix at all costs.

DR. A. R. CLAYTON said that in all probability surgeons in country districts saw appendicitis earlier than did hospital surgeons. It was the fixed rule at Moonta to operate immediately the condition was diagnosed, and practically every patient was operated on within twenty-four hours. It was particularly important to operate on children as early as possible. Either from an abundance of lymphatic tissue or a diminished resistance to the infecting organism, the appendices of children rapidly became very dangerous.

SIR HENRY NEWLAND congratulated Dr. Pomroy on the wisdom and understanding he had displayed in dealing with the subject. It was an easy matter to say that all cases of appendicitis should be operated on forthwith and the appendix removed. It was easy to say that no case of appendicitis should be operated on. It often required wisdom and a nice judgement to say whether an operation should be performed, when it should be performed, and how it should be performed.

Sir Henry Newland thought that Dr. Pomroy's figures under-estimated the mortality of appendicitis. It was shown in the discussion on acute intestinal obstruction at the annual meeting of the British Medical Association in 1932 that there was a rising tide of deaths from acute intestinal obstruction following operations for appendicitis. Post-operative hernia in cases of appendicitis ending in suppuration was a not infrequent sequel. Much of the mortality and morbidity associated with appendicitis was due to lack of judgement and to faulty technique in the course of the operation. Problems connected with the treatment of appendicitis did not cease with the opening of the abdomen. There was still wide scope for the exercise of surgical wisdom.

DR. G. H. BURNELL wished to stress the importance of conservative treatment in the case of pelvic abscess. Even if such cases had to be drained, it was better to wait until the abscess was well developed, and more harm was likely to result from too early attempts to drain than would result from waiting. With regard to the site of drainage for pelvic abscess, he had had an interesting experience only a week or two before. He had prepared to drain a pelvic abscess *per vaginam*, but when the patient was on the operating table he had examined her *per rectum* and had been surprised to find how much closer the abscess was to the rectum than to the posterior fornix, with the result that he had ended by draining the abscess *per rectum*, with a satisfactory result.

He wished finally to point out that in cases of paralytic ileus following appendicitis great benefit was derived from a spinal anæsthetic, as by this means operation might sometimes be avoided altogether, or even if it was still necessary, it would be far easier to deal with a flaccid abdomen rather than to struggle with a protruding mass of distended gut. The spinal anæsthetic acted within a few minutes of its administration. The bowels had acted copiously in the three cases in which he had tried it.

DR. W. J. CLOSE congratulated Dr. Pomroy, but at the same time recognized that it was impossible to deal with all aspects of the problem in a short evening.

One feature of the subject that had been mentioned by Dr. Clayton was the treatment of children; these patients were usually considered an exception to the applicability of the Ochsner-Sherren delayed treatment, on account of the comparative slowness with which protective adhesions took place. Dr. Close personally would not hesitate to explore the abdomen of a child unless there were obvious signs of limitation of the process.

It seemed also that the treatment was more applicable to hospital patients, who were frequently not seen by the surgeon till after forty-eight hours from the onset of the attack, but as several general practitioners had mentioned, one was usually called in much earlier, and in Dr. Close's experience the cases were few which were not seen in private practice before the thirty-six hours had elapsed.

Referring to the choice of rectum or vagina for drainage, Dr. Close said that he had discovered a good many years ago that some gynæcologists preferred the rectum for the drainage of pelvic collections. As a house surgeon he had been requested by his honorary, the late Dr. Lynch, to open the *cul-de-sac* and insert a tube; later in the day the sister informed him that he had mistaken the canal and that the tube was in the rectum. To his relief, however, Dr. Lynch let him down lightly, with the assurance that it was probably the better method of attacking the trouble.

DR. PERCIVAL CHERRY thanked and congratulated Dr. Pomroy on his paper, and considered that it was necessary to reeducate the public to the need for delayed interference in the treatment of some cases of acute appendicitis. When he was a student at the Adelaide Hospital over twenty-five years ago, the teaching was to operate in almost every case, regardless of how long the patient had been ill. The result was that the public came to realize that an early operation was essential. This was borne out in general private practice. Thus in many cases when a patient took ill with abdominal pain, the diagnosis was presumed by the patient or his relatives before the doctor was summoned; the medical man was sent for to come as soon as possible, because the patient had appendicitis and would need immediate surgical treatment. Often, of course, the patient was mistaken, as the condition was not appendicitis. If the signs and symptoms were indefinite and it took a few days to make the diagnosis, the patient might still insist that an early operation was essential, so it would be necessary to educate the public that there was a definite danger period, and operation, if not done soon after the attack occurred, should be delayed until that danger period had passed. There was nothing really new in this treatment as outlined by Dr. Pomroy, as fifteen years ago, when working with

Dr. Hone and Dr. Magarey, Dr. Cherry had discussed a certain case as to whether it would be safer to wait a few days before operating. Some years ago Dr. Cherry had met Dr. Sherren (after his retirement) on a mail-boat, and was astounded when Dr. Sherren said that he would never operate on a case of suppurative appendicitis under a week. When asked what he would do, Dr. Sherren said: "Keep in bed on starvation and operate when things had quietened down."

Dr. Cherry said that it was easier to keep patients in bed under efficient observation in hospital practice than it was in private practice. In the latter early operation was the usual procedure, and was more or less demanded by the patient, and would continue to be so until the public was educated to the fact that a danger period existed. Too often patients had been told that the delay was dangerous, whereas the real danger was that the delay had not been long enough.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warrnambool on July 22, 1933. Part of the report of this meeting has been published in the issue of December 23, 1933.

Lymphatic Leuchæmia.

DR. EDWARD BANNON showed a male patient, aged sixty-five years, who on April 18, 1933, complained of loss of appetite and a feeling of discomfort in the upper part of the abdomen for the previous two weeks. He felt as if his abdomen was swollen after meals, and he had a feeling of nausea and vomited on one occasion. There was no pain. For the past three months he had gradually been becoming more constipated, and he had two attacks of diarrhœa in that period, and had lost 4·5 kilograms (ten pounds) in weight. He also complained of feeling giddy at times and of shortness of breath on exertion. There were no other symptoms.

Examination showed a thin, elderly man, apparently wasted, and with a pale muddy complexion. The throat and mouth were clear, the chest was emphysematous, and examination of the heart and lungs revealed no abnormalities. The skin of the abdomen was loose and there was an inguinal hernia on the right side. Palpation revealed a large mass in the upper part of the abdomen. It was situated somewhat towards the left side and extended from the costal margin to the level of the umbilicus. It was very hard and immovable. Its upper margin could not be palpated, but the lower and left margins could be made out, and it appeared to shelve steeply posteriorly. It was dull on percussion. The liver was not enlarged, and the spleen was not palpable.

There were no enlarged supraclavicular glands, but there were three enlarged glands in the right axilla. These glands were small, discrete, very hard and freely movable. No other enlarged glands were detected. Rectal examination revealed no abnormality.

Barium meal examination showed that the œsophagus, stomach and duodenum were apparently normal. A barium enema showed that the rectum and ascending colon filled well, but there was a delay at the splenic flexure. A film showed that the transverse colon did not contain much barium, but sufficient to show that its contour was normal; it appeared to be flattened out. The descending and upper part of the ascending colon were well filled and appeared normal.

X ray examination of the spine at the level of the mass revealed no abnormality except some arthritic changes. X ray examination of the chest revealed that the lungs appeared normal, but suggested some enlargement of the mediastinal glands.

Blood examination was carried out and revealed the following:

Red cells, per cubic millimetre	3,840,000
Hæmoglobin value	60%
Colour index	0·8
White cells, per cubic millimetre	71,500
Neutrophile cells	13·5%
Eosinophile cells	nil
Basophile cells	0·2%
Lymphocytes	80·3%
Transitional cells	1·3%
Large mononuclear cells	0·7%

Films showed slight anisocytosis and poikilocytosis. There was no polychromasia or punctate basophilia, nor were any nucleated red cells seen. Lymphocytes were present in large numbers and were mostly of a large type.

On May 23, 1933, both legs began to swell, and in a few days the œdema was considerable.

On July 2, 1933, a course of deep X ray therapy was commenced in Melbourne and was continued for some weeks. The axillary glands disappeared almost entirely after the first treatment and the œdema of the legs disappeared soon afterwards.

On July 20, 1933, the patient was very well. The abdominal mass was still palpable, but much smaller.

Blood examination on this date revealed:

Red cells, per cubic millimetre	4,720,000
Hæmoglobin value	75%
Colour index	0·8
White cells, per cubic millimetre	26,000

The films showed slight anisocytosis and poikilocytosis, but nothing else abnormal, except a preponderance of lymphocytes.

DR. S. O. COWEN said that the point of greatest doubt and interest was the nature of the abdominal tumour. He thought that it was almost certainly a mass of recto-peritoneal glands, from its shape and from the signs of pressure on the *vena cava*, although it might possibly have been a lymphatic infiltration of the organs in the portal area. This patient presented a type of lymphatic leuchæmia in which, in Dr. Cowen's experience, the prognosis was exceptionally good with X ray treatment. One patient he knew at present was alive and comparatively well after eight years' treatment—an experience which was contrary to the usual text book teaching.

DR. LESLIE HURLEY said he considered there was little doubt as to the diagnosis. As to prognosis, text book statistics indicated that this was better in the myeloid than in the lymphatic type, but in the case of lymphatic leuchæmia with an increase of small lymphocytes only the prognosis was better than in the myeloid type. In the lymphatic type with larger and immature cells the prognosis was very bad, and the condition was usually made worse with X rays. It was rare to see such a patient with a fully developed blood picture without a palpable spleen. Dr. Hurley thought that œdema of the ankles with an anæmia showing only 60% hæmoglobin did not necessarily indicate pressure on the veneus return.

DR. JOHN HAYDEN said that the majority of cases of acute lymphatic leuchæmia were probably really myeloblastic leuchæmia. If these could be excluded from the statistics, then the chronic lymphatic leuchæmia would have a relatively good prognosis. He considered that the outlook in the present case was good for several years.

Dr. Bannon said that the epigastric mass had been three times its present size and he had no doubt that it was a mass of enlarged glands, although there was an empty feeling in the region of the spleen. When the patient was first seen there were enlarged glands in his axilla, which had been noticed for at least two years before, so that the patient had apparently survived already for two years without any treatment, and he could be expected to respond further to X ray treatment.

Endocarditis.

DR. IRVING BUZZARD showed a boy, aged thirteen years, who was first seen on July 1, 1932. The history was that a week before he had a fight with a boy going to school, in which he came off worse. He ran home to his mother very white and agitated and short of breath. His mother kept him in bed till he was seen by Dr. Buzzard.

On examination the boy's temperature was normal, his pulse rate was 124 and the respiratory rate 36. The apex beat was 12·5 centimetres (five inches) from the mid-line, in the sixth intercostal space. There was one and a half fingers' breadth of right cardiac dulness. A gross systolic murmur was heard all over the chest, with a marked diastolic murmur equally audible. No pericarditis was present. The patient had no fever. No pulmonary œdema or consolidation was found. The liver was not enlarged. The child was very distressed on the least exertion.

The past history was that eight years ago the child had influenza and rheumatism and was kept in bed for four to six weeks. After he got up his knees swelled and he was treated by his mother with home remedies. He was not ill again until he came under observation in July, 1932, though the mother noticed that he got short of breath easily on exertion.

The patient was kept in bed till March, 1933 (eight and a half months). During that time his heart rate was controlled with digitalis. The heart became normal in size, the murmurs disappeared, except for an apical systolic murmur conducted out into the axilla. The patient was then allowed graduated exercises.

He was very well till May 21, 1933, when he got a severe fright. His mother put him to bed and during the next seven days noticed that he was feverish and that he had two "fainting turns".

He was admitted to hospital on May 28, 1933. On examination his temperature was 38·5° C. (101·4° F.), his pulse rate 130 and his respiratory rate 38. The apex beat was in the sixth intercostal space, 13·75 centimetres (five and a half inches) from the mid-line. There was two fingers' breadth of right cardiac dulness. Systolic and diastolic murmurs were heard over all the valvular areas. No precordial rub was audible. The lungs were clear. The liver was not enlarged. The urine was clear. He was given salicylate of soda and digitalis.

The patient looked and remained very toxic. The temperature remained about 38·3° C. (101° F.), with a diurnal variation of about 1° C. The pulse rate came down to 80 or 90 with treatment, but the respiratory rate remained high. The heart condition was stationary. At the end of three weeks Dr. Bannon reported that *Staphylococcus albus* was obtained on blood culture. The patient was then given four injections of staphylococcus "Immunogen", 0·24 mil (four minims) and daily increases by the original dosage were made. There was no marked change in the patient's chart, though he certainly looked a little less toxic. After an interval of three days the "Immunogen" was repeated in the same doses. There was then much improvement in his general condition. On July 18, 1933, the heart was still grossly enlarged, but less enlarged than on his admission. There were marked systolic and diastolic murmurs at both mitral and aortic valves.

Dislocation of the Clavicle.

Dr. Buzzard also showed a male patient, aged twenty-one years, by occupation an iron moulder. On July 8, 1933, whilst playing football, he fell on to the point of his left shoulder. Movement was much restricted and he had to leave the field. Examination revealed a dislocation of the acromial end of the clavicle. The dislocation was easily reduced without anæsthesia. A pad of strapping was placed over the acromio-clavicular joint and was kept in position by a strip of plaster with the elbow and forearm supported. X ray examination nine days later showed the position of the bones to be good.

Arthritis of the Right Knee in a Case of Paget's Disease (Osteitis Deformans).

DR. A. E. BRAUER showed a male patient, aged fifty-nine years, who in 1922 first noticed that his right knee was larger than the left and less free in movement. Both these conditions gradually progressed and other joints became involved to a less extent. The patient's left leg was injured when he was in a ship that was torpedoed in 1915. At that time he had a severe attack of dysentery. He had trench fever in France in May, 1917; he was off duty for six weeks, had a recurrence in July, and was sent to England, where he again had an attack of dysentery.

Five weeks before the meeting the patient fell when his right knee gave way under him and he knocked his knee against a stone. The knee was very bruised and swollen. Fluid was present in the joint and a hæmatoma which formed over the knee was aspirated and the swelling then subsided fairly rapidly. A skiagram of the knee showed a fractured patella and a thickening of the bone, which was very porous. The other knee showed a similar porosity of both the femur and tibia.

On further examination the patient was seen to be markedly kyphotic, the head and humerus were enlarged, and he walked slightly bow-legged.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Mater Misericordiæ Public Hospital, South Brisbane, on October 6, 1933. The meeting took the form of a series of demonstrations by the members of the honorary staff.

Thrombosis of the Lateral Sinus.

DR. H. V. FOXTON showed a girl, aged twelve years, who, at the age of three years, was said to have had in the right ear a lemon seed, which was supposed to have been removed. From that time onwards she gave a history of having had occasional attacks of otorrhœa, but she did not complain of any pain. There would be slight discharge from the ear if she had a cold. On July 25, 1933, she had developed earache; about one week later the right ear commenced discharging, but there had been no ache whatever. On August 14 the temperature was taken for the first time and was found to be 41·1° C. (106° F.) and the patient had a rigor. She had been taken to Maryborough by train and was there seen by a medical man; she had several rigors and the temperature reached 40·5° C. (105° F.). On August 17 she arrived in Brisbane. On examination Dr. Foxton found that there was no tympanic membrane at all on the right side; there was no œdema over the mastoid area; the patient had a yellow septic appearance and was wasted. Although there was no definite mastoid tenderness, there was a slight suspicion of tenderness over the anterior process and slight tenderness on the right side of the neck. Pus was discharging from the right ear; there was no hearing at all in the ear. On the same day, August 17, a radical mastoid operation was done on the right side; the bone was found to be hyperæmic, with streaks of greyish pus in it. The question arose whether in a child of twelve years inflammation of the mastoid area alone would have caused such acute illness, but as the lateral sinus appeared to be normal, Dr. Foxton decided to wait a few days and see whether her condition would settle down. The temperature fell the day after operation; on the third day the patient had a rigor, and on the fourth day the lateral sinus was opened. It was found to be diminished in its lower part. The internal jugular vein was of very small calibre, no larger than a normal facial vein, and 6·25 centimetres (two and a half inches) of it had to be cut out. The facial vein also was reduced in size. The temperature subsequently rose twice to 40° C. (104° F.) and then gradually fell. The failure of the temperature to subside was probably due to the fact that the wound in the neck suppurated.

Dr. Foxton said that it was interesting to see how very insidious the progress of these conditions could be. This patient had had no pain, practically no tenderness, and only a high septic temperature and rigors to denote how ill she was.

Tic Douloureux.

DR. E. D. AHERN showed a male patient, aged fifty-three years, who had complained that for the last ten years he had suffered from attacks of acute spasmodic pain on the left side of the face. Sometimes over a period of twelve months he would have an attack about every six weeks, the attack varying slightly in intensity. Cold weather made the attack worse. Sometimes the attack would occur in the months of May and November, corresponding roughly to autumn and spring, but as the man's memory

was not accurate, these facts could not be definitely correlated. One thing, however, was definite, that the cold weather always made the attacks worse. When the patient was seen, his face was wrapped in flannel and he looked pale and ill. He stated that he had had nothing to eat for some days and shaved with difficulty. The spasm was over the left side of the face and extended from the eye to the upper lip. Operation was performed on July 11, 1933, the sensory root of the trigeminal nerve being destroyed. The sensory root behind the ganglion was an area with no power of regeneration, and section of this led to absolute and permanent removal of sensation of pain on that side of the face. Section of the root behind the ganglion also was not half so productive of trophic effects as removal of the ganglion. The eye had to be watched very carefully, as if any grit entered it a perforating ulcer might appear. The eye was kept closed after the operation and "Argyrol" was instilled regularly, and after three months any fear of trophic disturbance passed off.

Dr. Ahern wished to emphasize several points about the operation: (i) the position of the incision, (ii) the need for hæmostasis and the use of suckers, (iii) the importance of reaching the level of the skull before touching the ganglion, (iv) the blocking of the *foramen spinosum* with some material such as sterile gutta-percha, (v) the need for saving the motor root of the ganglion by lifting it off with a strabismus hook.

Injuries to the Wrist Joint.

DR. G. W. MACARTNEY showed a series of cases illustrating injuries to the wrist joint.

The first patient was a boy who, while riding a bicycle, ran into a fence and injured his wrist. An X ray examination revealed a fracture of the scaphoid bone and a dislocation of the semilunar bone, which stood right out from the carpus. The boy was given an anæsthetic and very strong extension was necessary to get the bone back into position. A pad and a broad band were put round the arm, which was held by a nurse; the hand was painted with glue and two thicknesses of gauze were then applied. This prevented slipping of the hand when manipulation was tried, and also protected the skin. The nurse pulled on the arm and Dr. Macartney manipulated the wrist, leaving the little finger out of his grasp, and reduced the deformity to a good position, as was shown by X ray examination. This had been done six weeks previously, and the hand and wrist were put into a non-padded plaster cast, the volar part being especially well moulded so that it would not slip. Four days ago the plaster had been removed; there was good movement at the wrist and there had been no pain while the hand was in plaster, and yet the fracture of the scaphoid was still very pronounced, with quite a degree of separation. The hand was put up in plaster again and eventually it would unite. This case brought up the point that excision of the scaphoid was considered to be wrong, as the bone would unite in time.

Dr. Macartney's second patient was a boy who was suffering from an old injury to the wrist. Six years previously he had fallen seven feet down a bank and had sprained his wrist. He had returned to work in a few days and had worked continually since in a foundry. He now noticed that with heavy jarring his wrist became painful. X ray examination showed a huge projection on the semilunar bone. The report stated that there had been a fracture dislocation of the semilunar bone and that *caries siccum* was present in that bone. The lunate bone had been partly dislocated on the *os magnum* and arthritis was present in that joint. The question of treatment arose. Should the lunate bone be removed? There was some limitation of dorsiflexion at the wrist and some thickening.

Dr. Macartney's next patient was a man who had injured his wrist six weeks previously while cranking a motor car. The hand had been put on a splint and the patient sent to hospital for diathermy treatment. The wrist was not examined by X rays till the patient went to the out-patient department on September 29. The X ray examination revealed periostitis over the scaphoid side of the semilunar bone, but no fracture. The patient had been wearing a straight metal splint, which was taken off each day while diathermy treatment was given. Disability was still present and pain prevented him from working. Dr. Macartney considered that the hand should be put in plaster for six weeks at least and then tested for disability. This lesion was after the type of early Kienbock's disease; the results of treatment were not very satisfactory, especially if the condition were advanced.

Pick's Disease.

DR. ELLIS MURPHY showed a female patient, aged thirty years, who was interesting in that to October, 1932, she had been quite well. She gave no history of rheumatic fever, growing pains or chorea. One evening in October, 1932, she had had an acute attack of dyspepsia; one hour later her feet had swelled. She was seen by a medical practitioner and sent into hospital for two weeks, and then had been in bed at home for four weeks. She had been fairly well for six or eight weeks, though never quite free from œdema of the legs, and for the last four months she had had progressive swelling of the abdomen and dyspnœa.

When Dr. Murphy saw her, she had œdema of the legs and of the back up to the level of the scapula, with marked ascites and a right pleural effusion. The heart was definitely displaced to the left and apparently enlarged, and there was some retraction of the precordium during systole. The patient was admitted to hospital on September 5 and "Salyrgan" was given every three days. One hundred and eighty cubic centimetres (six ounces) of urine were passed the first day, 2·58 litres (eighty-six ounces) after one cubic centimetre of "Salyrgan". The following day only 300 cubic centimetres (ten ounces) of urine were passed, and the next day 210 cubic centimetres (seven ounces). Another dose of "Salyrgan" was therefore given, and 3·72 litres (124 ounces) of urine were passed. "Salyrgan" was therefore given every third day over a period of fourteen days. The pleural cavity was tapped and approximately 1·8 litres (60 ounces) of fluid were removed every few days, the last being on September 30.

Bronchiectasis.

Dr. Murphy's second patient was a girl, aged twelve years, whose mother complained that she had had a cough since she was eight months old. The child had never been strong, had suffered from frequent colds, and the cough had become very much worse in the last few years. The girl herself complained of headaches. Examination revealed a well developed girl, whose colour was good. There was no clubbing of the fingers. Examination of the chest revealed coarse medium râles over the lower two-thirds of both lungs. A diagnosis of bronchiectasis of a mild type was made. An X ray examination was made with lipiodol in both lungs. On the right side the bronchi showed up fairly well and there was very little dilatation. On the left side there was lack of filling and atelectasis in the area behind the heart (the most common region) and the bronchi were dilated there. An X ray picture of the antra showed that these were almost obliterated with thickened mucosa and polypoid material.

Hemianopia.

Dr. Murphy's next patient was a man, aged forty years, who was suffering from hemianopia. Four or five weeks previously he had had influenza with a severe cough. One day, after coughing, he walked across a room and fell over a chair and a child; he then found he could not see on his left side. Examination revealed a left-sided hemianopia with retention of the fixation point, and a diagnosis was made of a thrombosis of the calcarine branch of the occipital artery on the right side. The blood pressure was normal, the Wassermann test gave no reaction. The antra were found to be infected. Dr. Hoare had reported that there was no retinal artery sclerosis. The patient had improved in the last two weeks. The macula was retained and he did not get complete blindness of the fixation point; this pointed probably to a vascular lesion. At the time of the meeting the field of vision was almost normal. There were no other changes present, and probably the area of the lesion was very far back in the optic tract.

Case for Diagnosis.

DR. P. N. MACGREGOR presented a male patient for diagnosis, which, however, had been practically settled by an X ray examination made that day. A man, aged fifty-eight years, had complained of indigestion, by which he meant fullness and heartburn, for thirty years. He had had no vomiting and no actual pain and the condition was relieved by taking bicarbonate of soda. He would have a definite attack for some weeks and then would have relief. Four or five years previously he had had pain on swallowing, then pain a half to one hour after his meals, this latter being occasionally relieved by bicarbonate of soda. At that time he vomited blood and the motions were tarry. He was short of breath on exertion and complained of pain over the cardiac area, radiating to the upper part of the left arm. This, however, had been absent for the last week. There was no history of venereal disease. He had lost between one and two stone in weight; he had no cough.

At present his complaint was weakness, shortness of breath, pain and difficulty in swallowing. He had been in hospital having two-hourly feeds together with alkaline powders.

On examination he was found to be anæmic and wasted. There was some epigastric tenderness, but no mass was felt. There was no enlargement of the liver or spleen. The heart was definitely enlarged to the left and especially to the right; the heart sounds were normal. A test meal showed some hyperacidity at the beginning; the fasting specimen contained some altered blood, as did all specimens. The Wassermann test gave no reaction. A blood film revealed 2,290,000 red cells per cubic millimetre, the hæmoglobin value was 46%, giving a colour index of 1·0. Anisocytosis and poikilocytosis were present. There were no nucleated red cells, and platelets were normal in number. An X ray examination made two months previously had shown no abnormality of the stomach or œsophagus, but another skiagram taken that day revealed that the condition was a carcinoma of the œsophagus.

Conservative Treatment of Peritonitis.

DR. J. C. HEMSLEY showed two patients who suffered from an acute infection in the peritoneal cavity. The condition was not an acute appendicitis, but there were indications that pus was present in each case, and conservative treatment was adopted.

The first patient was a married woman, aged thirty-five years, who was admitted to hospital with a history of pain for twenty-four hours in the lower part of the abdomen and in the right iliac fossa. She had a pulse rate of 140 per minute. She had a toxic goitre, with enlargement of the gland and slight exophthalmos; the teeth were septic. She weighed just over 37·8 kilograms (six stone) and looked very ill. She admitted that she smoked a lot and had been taking alcohol. On examination of the abdomen the patient was found to have a definite general peritonitis in the lower part of the abdomen, with marked rigidity and tenderness up to the umbilicus. *Per vaginam* a large tender mass was felt filling the pelvis, and there was a profuse purulent discharge from the vagina. The patient was far too ill to be submitted to operation, so for four days she was given morphine and small sips of water. The bowels were not opened for eight days. The temperature dropped down quickly after fourteen days. *Per vaginam* examination then showed that the whole condition had subsided, so the patient was sent home on a diet of milk and plain food. Two months later she had two to three weeks in bed, then her goitre was removed and she was doing well.

From her history it was concluded that the condition was a gonorrhœal infection, of a type that settled down. The important point in the treatment was absolute rest.

The second patient was a woman who looked very ill and who had a profound secondary anæmia. She had a purulent discharge from the urethra and vagina, with an acute vaginitis, and *per vaginam* a large tender mass was felt filling the pelvis; tenderness extended up to the umbilicus. A diagnosis was made of acute salpingitis with pelvic peritonitis, but as the patient was too ill for operation conservative measures were used. The condition improved, though at first there was no marked fall in temperature; however, after ten days it dropped to normal. Treatment was along the same lines as those adopted with the previous patient, namely, starvation and morphine. At the end of six weeks the condition had apparently subsided sufficiently for operation, but it was found that the adhesions had not become organized sufficiently to enable Dr. Hemsley to carry out extensive dissection. The patient had vomited after operation and the temperature had risen for three or four days, but it eventually subsided.

Dr. Hemsley considered that conservative treatment could certainly always be used for localized infections in the pelvic cavity and frequently in cases of acute appendicitis. He did not say that early operation should never be carried out, but that careful watch should be kept. There was plenty of time. The treatment should be thorough and the conservatism complete; there should be no interference with the bowels, and no food should be given.

SCHOLARSHIPS AND GRANTS IN AID OF SCIENTIFIC RESEARCH.

Scholarships.

THE Council of the British Medical Association is prepared to receive applications for research scholarships as follows:

An Ernest Hart Memorial Scholarship, of the value of £200 *per annum*.

A Walter Dixon Scholarship, of the value of £200 *per annum*.

Three research scholarships, each of the value of £150 *per annum*.

These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including state medicine) relating to the causation, prevention or treatment of disease.

Each scholarship is tenable for one year, commencing on October 1, 1934. A scholar may be reappointed for not more than two additional terms. A scholar is not necessarily required to devote the whole of his or her time to the work of research, but may hold a junior appointment at a university, medical school or hospital, provided the duties of such appointment do not interfere with his or her work as a scholar.

Grants.

The Council of the British Medical Association is also prepared to receive applications for grants for the assistance of research into the causation, treatment or prevention of disease. Preference will be given, other things being equal, to members of the medical profession and to applicants who propose as subjects of investigation problems directly related to practical medicine.

Conditions of Award: Applications.

A copy of the regulations relative to the award of the scholarships and grants for 1934, and of the prescribed application form can be obtained on application to the Secretary of the Federal Council of the British Medical Association in Australia, British Medical Association House, 135, Macquarie Street, Sydney. The completed application form is required to be submitted to the Secretary of the Federal Council not later than March 3, 1934.

Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated, to whom reference may be made.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on October 11, 1932, DR. F. KINGSLEY NORRIS, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Pseudosclerema.

DR. J. W. GRIEVE showed a baby of six weeks who had been admitted to the ward at the age of five days because of an indurated rash on the buttocks which had commenced at the age of three days. This rash rapidly extended to other areas and was regarded as *erysipelas migrans*. During the entire stay of the child in hospital it was slightly febrile, the temperature varying between the normal and 38·3° C. (101° F.). It is stated that after the child left hospital there was a slight generalized fine desquamation, more especially over the areas of skin affected.

The child was discharged from the ward after three weeks, when the condition had subsided considerably, but several indurated areas remained on the back. One week later there was a recurrence of the condition on the back, the buttocks and the right calf, and in these areas it was still present.

The child had steadily increased in weight during the whole period of time it had been under observation. Physical examination revealed no other abnormality, and the family history was quite irrelevant to this condition.

Dr. Grieve drew attention to the classification of these diseases as outlined in Garrod Batten and Thursfield's "Diseases of Children". The diseases which were commonly confused are sclero-œdema, sclerema (including pseudosclerema) and sclerodermia. Briefly the distinguishing features were:

1. Sclero-œdema (*sclerema œdematosum*) occurred in premature and weakly or sometimes syphilitic babies. It occurred in the first week of life, usually the second to the fourth day, and affected the dorsum of the feet, the lower abdominal wall, the penis, scrotum and cheeks. The œdema was hard, it infiltrated the muscles, it pitted on pressure, it spread very rapidly and death occurred in a few days, with extreme collapse and a subnormal temperature.

2. Sclerodermia was rare in the first few weeks. It consisted of œdematous swellings progressing into a coarse thickening of the skin and subsequently into an atrophic condition. If it became general, the child was hidebound.

3. Sclerema was less common than sclero-œdema. It usually occurred in the first few days of life, but might occur at any time up to six months in weakly infants. It affected the parts richest in fat and not those which contained most subcutaneous loose cellular tissue. Commencing on the calves, it usually extended symmetrically to the thigh, trunk and neck, the head and upper extremities being last affected. The penis, scrotum, palms and soles where the fat was subfascial were not affected. The skin was flat, board-like, not raised, could not be picked up and would not pit. If the condition became generalized, the child was rigid. Death usually occurred in from two to eight days.

4. Pseudosclerema was more common. Here there were circumscribed areas of induration, often of a livid colour, with a sharply defined, raised, irregular edge. The patches did not pit. Usually the patches occurred on the buttocks, thighs and back. Recovery was the rule in anything from one to four months. Constitutional disturbance was not common. Radiography might reveal mottled opacities in the affected areas. Both sclerema and pseudosclerema were regarded as primary inflammations of the skin.

Dr. Grieve thought that the baby he presented was suffering from pseudosclerema.

Peroneal Muscular Atrophy.

Dr. Grieve next showed a boy of thirteen years who had developed difficulty in walking six years previously, apparently owing to inability to flex and evert his ankles. The weakness progressed in his lower limbs, especially in the muscles of the feet and legs. The weakness was accompanied by wasting. Two months ago weakness was noticed in each hand, and examination then revealed weakness and wasting of the intrinsic muscles of each hand. His family history was clear.

The boy's intelligence was normal. There was marked wasting of the intrinsic muscles of each hand, with weakness especially of the interosseous and lumbrical muscles. In these muscles there was no response to either faradism or galvanism, although there was a weak response in muscles below the elbow. In the lower limbs there was general wasting of muscles, especially below the knee, so that he could not evert or dorsiflex either ankle. There was very weak flexion and extension of the toes and also extension of the ankle only. There was no response to faradism or galvanism in the muscles below the knee. The deep and superficial reflexes were all present, the cranial nerves were all normal, and all forms of sensation were intact. There was no fibrillar twitching and no reaction of degeneration. The muscles of the face, trunk, and limb girdles were all normal. Dr. Grieve showed the boy as a sufferer from the neuritic form of progressive muscular atrophy of Charcot-Marie-Tooth, the strictly distal distribution distinguishing it from other forms of muscular atrophy. Progressive muscular atrophy usually commenced later in life, but it might start in childhood, so that age of onset alone was no criterion. But progressive muscular atrophy never kept strictly to the distal distribution. In the various forms of myopathy the weakness and wasting especially affected the face, the trunk, and the proximal muscles of the limbs. In this disease there was an atrophy of the anterior horn cells, with corresponding atrophy in the fibres of the peripheral nerves and of the muscles which they supply.

Recurrent Tetany.

DR. H. LAWRENCE STOKES showed a boy of fourteen years with a history that for the past four years he had suffered from recurring spasms of the hands and feet. He would have two or three of these carpo-pedal spasms each day for a week at a time and would then be free for some time. He had been given calcium and cod liver oil with varying effect. When Dr. Stokes saw him a month previously he was a typical sufferer from tetany, his blood calcium was 5·5 milligrammes per hundred cubic centimetres, and the blood phosphorus was 9·4 milligrammes. He was given calcium chloride, 0·6 gramme (ten grains), three times a day, but he was not given parathormone because recent work showed that though it mobilized calcium from the bones, it did not increase calcium absorption, and therefore would not help at all. Under this treatment his carpo-pedal spasms ceased, although both Trousseau's and Chvostek's signs could still be elicited with the appropriate stimulation. There was no apparent abnormality in either the thyreoid or parathyreoid glands. The skin, nails and enamel were all normal, there was no cataract, and radiographic examination of the bones revealed no abnormality. At the time of the meeting the figures for his blood calcium and phosphorus were 5·5 and 17·6 milligrammes per 100 cubic centimetres respectively, the phosphorus figure being the highest that Dr. Stokes had seen.

Dr. Stokes showed the boy as an example of recurrent tetany for which he could find no cause. Provided calcium was given in adequate amounts, his symptoms were easily controlled.

Correspondence.

MEDICAL EVIDENCE, CERTIFICATES AND FEES UNDER "THE WORKERS' COMPENSATION ACT".

SIR: I read with great interest the article on workers' compensation matters written by Dr. Mackay and published in your issue of December 9, 1933, and I consider it very comprehensive and sound, except the paragraph which begins as follows: "It is an indisputable fact that the provisions of the *Workers' Compensation Act* have diverted annually a large sum of money to the medical profession."

This sentence does not convey a true appreciation of the position at all, and personally I strongly resent it, especially coming from the pen of the "Chief Medical Referee and Medical Assessor to the Workers' Compensation Commission of New South Wales", and I hope it is not the official attitude of the learned judge who presides, and of other members of the Commission.

I submit, Sir, that a more just statement of the true position is conveyed by the following.

By the *Workers' Compensation Act*, 1926, and amendments the legislature of New South Wales has established two important principles: Firstly the principle that the medical man who attends a worker injured in the course of his employment is worthy of payment for his services, and secondly, that the worker has the right to be considered a self-respecting citizen, whose medical attention in the event of injury at work is provided by insurance and who is not compelled to ask for charity at the hands either of the medical profession or of those of his fellow citizens who contribute to public hospitals.

The medical provisions of the *Workers' Compensation Act* constitute a tardy act of justice to medical practitioners and allied professional workers, as well as to the actual workers engaged in industry.

Surely the public conscience in New South Wales has not sunk to so low a level that the remuneration paid to medical men for professional attention to injured workers is to be regarded as a "diversion" of money, especially as the insurers (who may be regarded as trustees of moneys received for this purpose) fix the premium rates and receive the hard cash before they undertake any contractual obligations to the employers.

Yours, etc.,

A. M. DAVIDSON.

Enmore,
December 14, 1933.

SIR: Section 10, Subsection 4*a* of the New South Wales *Workers' Compensation Act*, in referring to medical fees, says: "The sum for which an employer shall be liable in respect of the medical treatment of a worker shall be such sum as is reasonably appropriate to the treatment afforded, having regard to . . . . the customary charge made in the community for such treatment to persons other than workers." And the latter portion of Dr. R. M. Mackay's address (THE MEDICAL JOURNAL OF AUSTRALIA, December 9, 1933) is a defence of the Commission's ruling that "the customary charge should bear some relation to the worker's capacity to pay, *i.e.*, his earning capacity". I contend that the Act neither says nor intends anything of the sort. The wording is precise and definite, and its interpretation is a very vital matter in the present dispute between our Association and the Fire and Accident Underwriters' Association of New South Wales. The vital words in the clause are: "the customary charge made in the community for such treatment to persons other than workers."

The "customary charge" in the community in which I practise is 10s. 6d. a surgery visit and 12s. 6d. an outside visit to everybody, from "hoboes to millionaires"—a minimum rate decided by our local association and observed, I honestly believe, by all practising members. (In some few local associations the rates are respectively 7s. 6d. and 10s. 6d.) The fact that a proportion of our patients fail suitably to react to our "memoranda of fees due" or that from motives of private charity we render some gratuitous service does not alter the basic fact that the customary charge is 10s. 6d. *et cetera* to everybody. Because a grocer, from humanitarian motives, gives away free a pound of butter to some of his indigent customers, it does not follow that the customary charge for butter in the community is anything but the prevailing price, for example, 1s. 6d. per pound.

I venture to assert that in no doctor's records would it be possible to find entries of charges of, say, 1s. 6d., 4s., 6s. *et cetera*, depending on the patient's earning capacity, nor do I believe Dr. Mackay would be able to find a single instance of an insurance company paying higher rates than Schedule D because a worker happened to be earning, say, anything from £500 to £549.

Section 16 of the Act contains a fixed table setting out the lump sum compensation for certain specific injuries and in assessing the rates for loss of sight or limb awards the same rates to apprentices as managers, irrespective of their earning capacity, and I see no reason why an elastic scale can be read into the clause under dispute.

I presume that a doctor renders similar service to every class of injured worker, and as it is this service that is being paid for by the insurer, what has the patient's financial position to do with the matter? The doctor's service is aimed at restoring to function an injured and incapacitated member, and failure in this service renders the insurer liable for "lump sum" compensation according to the table in Section 16, irrespective of the worker's earning capacity. Why shouldn't the insurer pay equal rates for equal protection?

Dr. Mackay quotes some cases of apparently excessive charges by doctors. It is important to note here that in all these cases the aggrieved insurer has had access to a "referee tribunal" set up by our Association and which, I believe, has not hesitated, when necessary, to recommend reductions in doctors' accounts.

The case of the "fractured femur" in which £21 was charged was, in my opinion, a perfectly correct charge under Schedule D, and was perhaps one of the few cases which happen, to compensate for the other "flat rate" cases under Schedule D, as, for example, amputation of a septic finger joint, which may involve weeks of anxious treatment and for which the fee has been strictly interpreted by the insurer under Schedule D as four guineas.

Taking it by and wide, the schedule is a passable working scheme, but the experience of practising doctors has been that the incidence of the "septic finger" type of case has been a high multiple of the "fractured femur" type. I am sorry that I cannot share Dr. Mackay's sympathy for the insurance companies.

In discussing total compensation payments to injured workers, Dr. Mackay is shocked at the high ratio of medical costs to weekly compensation (sustenance) payments, namely, 13·31%. Reduced to simple figures, it simply amounts to this, that a worker so severely injured that he cannot work for at least seven days, finds that out of £5 sustenance he is receiving, 13s. 4d. is paid in medical expenses to repair his injuries. Surely his medical treatment is the most important part of his compensation. It is the means of shortening greatly the period of his incapacity and correspondingly curtailing his claims on the major cost of sustenance.

I can even conceive the possibility of circumstances where £37 spent on medical expenses and £39 on sustenance, as quoted in horror by Dr. Mackay, would be money equitably spent by an insurer.

In another case quoted by Dr. Mackay the patient had a septic leg requiring no less than fifteen weeks' hospital treatment. The doctor charged £49 for his services, and this on the facts may or may not have been excessive, but I am sure Dr. Mackay will not deny that without very constant, skilful medical treatment the worker's insurer could very conceivably be presented with a bill for from £525 to £600 for the loss of part or whole of the leg, and a greater amount for loss of life. This limb or life would be just as costly to the insurer and was valued

just as dearly by our legislators when framing the Act, even though the worker was earning only 30s. per week.

The spirit of the *Workers' Compensation Act* is one of humanitarianism and it was never intended to make distinctions in the treatment of various workers nor in the remuneration for such treatment.

In actual operation it has not been the statistical "gold mine" for the general run of practitioners that Dr. Mackay thinks. I feel sure that in many cases the trouble, loss of temper, and roundabout formalities in having just claims settled have so disheartened doctors that many have often felt like declining to take workers' compensation cases. At most, it has, in a proportion of cases only, secured to a medical attendant his fair and just dues, namely, payment for honest services rendered.

Yours, etc.,

RICHARD D. DAVEY.

Croydon Park,
New South Wales,
December 13, 1933.

Books Received.

INFECTIONS OF THE HAND: A GUIDE TO THE SURGICAL TREATMENT OF ACUTE AND CHRONIC SUPPURATIVE PROCESSES IN THE FINGERS, HAND AND FOREARM, by A. B. Kanavel, M.D., Sc.D.; Sixth Edition, thoroughly revised; 1933. Philadelphia: Lea and Febiger. Royal 8vo., pp. 567, with 216 engravings.

MUSIC AND THE LISTENER: A GUIDE TO MUSICAL UNDERSTANDING, by K. Barry, M.B., Ch.M.; 1933. Melbourne: Robertson and Mullens, Limited. Crown 8vo., pp. 126. Price: 2s. 6d. net.

CHRONIC NEPHRITIS AND LEAD POISONING, by L. J. J. Nye, M.B., Ch.M.; 1933. Australia: Angus and Robertson, Limited. Demy 8vo., pp. 163, with illustrations. Price: 12s. 6d. net.

A SYSTEM OF CLINICAL MEDICINE DEALING WITH THE DIAGNOSIS, PROGNOSIS AND TREATMENT OF DISEASE FOR STUDENTS AND PRACTITIONERS, by T. D. Savill, M.D.; edited by A. Savill and E. C. Warner, M.D.; Ninth Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 1093, with illustrations. Price: 28s. net.

THE PRACTICE OF SURGERY, by R. Howard, C.B.E., M.S., F.R.C.S., and A. Perry, M.S., F.R.C.S.; Fourth Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 1345, with eight coloured plates and 584 illustrations in the text. Price: 30s. net.

MR. PUNCH AMONG THE DOCTORS; 1933. London: Methuen and Company, Limited. Demy 4to., pp. 104. Price: 5s. net.

ORGANIC AND BIO-CHEMISTRY, by R. H. A. Plimmer, D.Sc.; Fifth Edition; 1933. London: Longmans, Green and Company. Royal 8vo., pp. 634, with illustrations. Price: 21s. net.

THE NATURE AND TREATMENT OF AMENTIA: PSYCHO-ANALYSIS AND MENTAL ARREST IN RELATION TO THE SCIENCE OF INTELLIGENCE, by L. P. Clark, assisted by the staff of the Psychoanalytic Sanatorium at Rye, N.Y., with foreword by E. Jones, M.D.; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 322. Price: 12s. 6d. net.

HEREDITY AND THE SOCIAL PROBLEM GROUP, by E. J. Lidbetter; Volume I; 1933. London: Edward Arnold and Company. Crown 4to., pp. 160, with charts. Price: 21s. net.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xiv.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Medical Superintendent (male).

THE AUSTRALIAN INLAND MISSION, SYDNEY, NEW SOUTH WALES: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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